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Gas Exchange During Sleep in Patients on LTOT

New Technologies for Lighter Portable Oxygen Systems

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Predicting Nighttime Gas Exchange Using Daytime Arterial Blood Gases in Patients on LTOT

Maintaining Oxygenation via Demand Oxygen Delivery During Rest and Exercise

Impact of a Radio Frequency Management Information System on an Inpatient Respiratory Care Service

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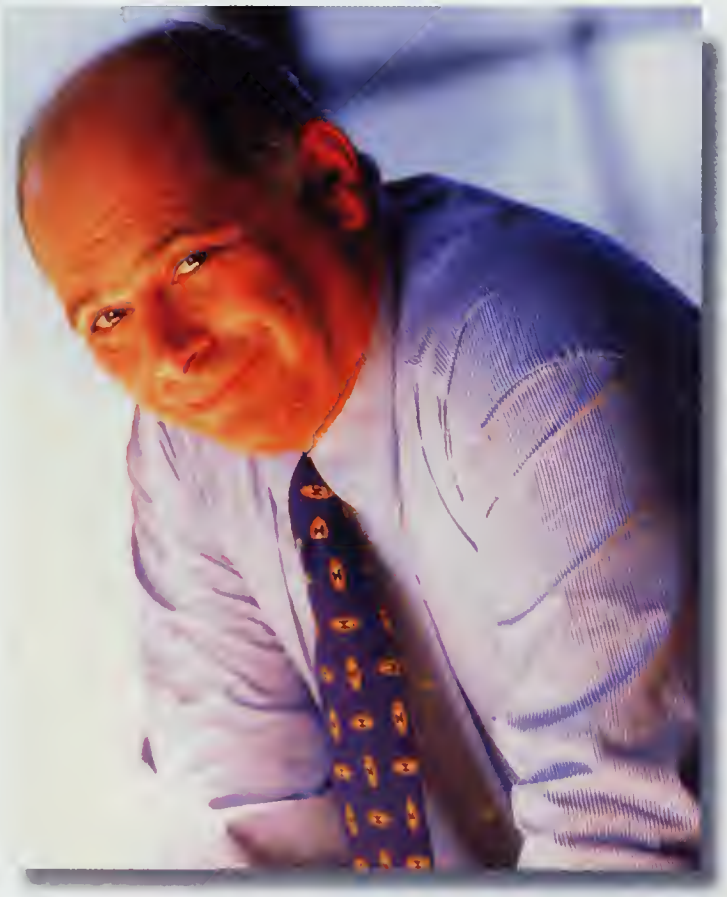

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The Wendland case—Withdrawing Life Support from Incompetent Patients Who Are Not Terminally Ill—Lo B, Dornbrand L, Wolf LE, Groman M. N Engl J Med 2002 May 9;346(19):1489-1493.

Bronchiectasis—Barker AF. N Engl J Med 2002 May 2;346(18):1383-1393.

From the Centers for Disease Control and Prevention. Annual Smoking Attributable Mortality, Years of Potential Life Lost and Economic Costs—United States, 1995-1999. JAMA 2002 May 8;287(18):2355-2356.

Transition in Cystic Fibrosis: Much Ado About Nothing? A Pediatrician's View—Schidlow DV. Pediatr Pulmonol 2002 May;33(5):325-326.

Sleeping with the Enemy: The Heart in Obstructive Sleep Apnea (editorial)—Pinsky MR. Chest 2002 Apr;121(4):1022-1024.

Selecting the Right Level of Positive End-Expiratory Pressure in Patients with Acute Respiratory Distress Syndrome—Rouby JJ, Lu Q, Goldstein I. Am J Respir Crit Care Med 2002 Apr 15;165(8):1182-1186.

Nitric Oxide, Phosphodiesterase Inhibition, and Adaption to Hypoxic Conditions—Wilkins MR, Aldashev A, Morrell NW. Lancet 2002 May 4;359(9317):1539-1540.

Functional Hemodynamic Monitoring—Pinsky MR. Intensive Care Med 2002 Apr;28(4):386-388.

Intra-Abdominal Pressure: Time for Clinical Practice Guidelines?—Sugrue M. Intensive Care Med 2002 Apr;28(4):389-391.

Indoor Swimming Pools, Water Chlorination and Respiratory Health (editorial)—Nemery B, Hoet PH, Nowak D. Eur Respir J 2002 May;19(5):790-793.

Paediatric Asthma—A New Virtual Journal (editorial)—Marcovitch H, Williamson A. BMJ 2002 May 4;324(7345):1052.

Pulmonary Alveolar Proteinosis: Step-by-Step Perioperative Care of Whole Lung Lavage Procedure—Ben-Abraham R, Greenfeld A, Rozenman J, Ben-Dov I. Heart Lung 2002 Jan-Feb;31(1):43-49.

BACKGROUND: Pulmonary alveolar proteinosis is a rare disease characterized by the accumulation of surfactant-like material within the alveolar spaces that causes progressive respiratory failure. Improvement can be achieved with whole lung lavage. OBJECTIVE: Our objective was to conduct a study of the feasibility of treating pulmonary alveolar proteinosis in a community hospital. METHODS: Five patients were treated. We assessed procedure pulmonary functions. RESULTS: No major sequelae occurred. Each lung was lavaged with 12 to 20 L of normal saline in cycles of 970 ± 150 mL each (mean ± standard deviation), over 106 ± 49 minutes. Extubation was performed when compliance of the lavaged lung was restored. All patients showed subjective improvement. Resting and exercise oxygen saturation improved within 1 week after the lavage. A significant improvement was also noted in forced expiratory volume in 1 second, forced vital capacity, and maximal oxygen uptake, whereas total lung capacity and carbon monoxide single-breath diffusion capacity remained unchanged. CONCLUSION: Although retrospective and based on a small sample size, our results suggest that whole lung lavage may be performed safely even in medical centers that have limited experience, if strict adherence to a protocol is maintained.

The Modified Nasal Trumpet Maneuver—Beattie C. Anesth Analg 2002 Feb;94(2):467-469.

The last decade has witnessed a proliferation of devices or methods that facilitate intubation in difficult circumstances, maintain ventilation, or which do both. These all require properly functioning and specially designed apparatus, the use of which requires variable degrees of expertise. This technical communication describes the author's experience with a simple technique that uses virtually universally available materials—a nasal trumpet (airway) and an endotracheal tube (ETT) connector—to rescue patients in the cannot-ventilate/cannot-intubate scenario. The methodology is straightforward, ventilation is usually immediate, stomach contents can be evacuated while ventilation proceeds, and it does not require mouth opening. Moreover, while ventilation and oxygenation is continuing, a fiber-optic intubation can proceed without interference. IMPLICATIONS: A simple technique is proposed that can be used to rescue patients who are in a condition of cannot intubate/cannot ventilate. The described maneuver may save patients from requiring a surgical airway.

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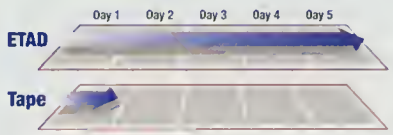

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1. Clinical evaluation of an Oral Endotracheal Tube Attachment Device (ETAD) Hollister, 1995.
2. Cunneen, Jane A different approach to endotracheal tube stabilization Hollister, 1996.

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The Early Course of Newly Diagnosed Asthma—Ernst P, Cai B, Blais L, Suissa S. Am J Med 2002 Jan;112(1):44-48.

PURPOSE: We describe the intensity of therapy for patients with newly diagnosed asthma and how it changed during subsequent years in relation to age, sex, and initial level of therapy. METHODS: We examined a cohort of 13,671 patients in Saskatchewan, Canada, who were initially between the ages of 5 and 44 years. Patients were followed prospectively, and the intensity of asthma therapy was measured during successive 12-month periods. RESULTS: Based on the intensity of asthma drug therapy during the first year after entry into the cohort, 6661 patients (48.7%) were initially prescribed therapy judged to be appropriate for mild asthma, and 977 (7.1%) were dispensed medications in a manner suggesting their asthma was severe; the remaining 6033 (44.1%) were classified as receiving treatment of intermediate intensity. Among patients initially classified as receiving treatment appropriate for mild disease, only about 3% were dispensed medications that suggested that their asthma had become severe during up to 5 years of follow-up. Intensity of therapy waned in a substantial proportion of patients who were initially classified as having severe asthma, especially if they were initially younger than 15 years of age. Thirty-four per 100 patients initially younger than 15 years old were receiving medications appropriate for mild asthma, and 23 per 100 such patients received no medication for asthma during a 12-month period when followed up to 5 years. CONCLUSION: Patients with asthma who are initially treated with therapy appropriate for mild asthma are rarely treated later with therapy suggesting the advent of severe disease. Patients initially dispensed medications suggesting the presence of severe asthma often see the intensity of treatment wane over time.

Similar Ventilation Distribution in Normal Subjects Prone and Supine During Tidal Breathing—Rodriguez-Nieto MJ, Peces-Barba G, Gonzalez Mangado N, Paiva M, Verbanck S. J Appl Physiol 2002 Feb;92(2):622-626.

Multiple-breath washout (MBW) tests, with end-expiratory lung volume at functional residual capacity (FRC) and 90% O_2, 5% He, and 5% SF_6 as an inspired gas mixture, were performed in healthy volunteers in supine and prone postures. The semilog plot of MBW N_2 concentrations was evaluated in terms of its curvilinearity. The MBW N_2 normalized slope analysis yielded indexes of acinar and conductive ventilation heterogeneity (Verbanck S, Schuermans D, Van Muylem A, Paiva M, Noppen M, and Vincken W. J App Physiol 83: 1907-1916, 1997). Also, the difference between SF_6 and He normalized phase III slopes was computed in the first MBW expiration. Only MBW tests with similar FRC in the prone and supine postures ($p > 0.1$; $n = 8$) were considered. Prone and supine postures did not reveal any significant differences in curvilinearity, N_2 normalized slope-derived indexes of conductive or acinar ventilation heterogeneity, nor SF_6-He normalized phase III slope difference in the first MBW expiration ($p > 0.1$ for all). The absence of significant changes in any of the MBW indexes suggests that ventilation heterogeneity is similar in the supine and prone postures of normal subjects breathing near FRC.

Effect of Gravity on Chest Wall Mechanics—Bettinelli D, Kays C, Bailliart O, Capderou A, Techoueyres P, Lachaud JL, et al. J Appl Physiol 2002 Feb;92(2):709-716.

Chest wall mechanics was studied in four subjects on changing gravity in the craniocaudal direction (G_z) during parabolic flights. The thorax appears very compliant at 0 G_z: its recoil changes only from -2 to 2 cm H_2O in the volume range of 30-70% vital capacity (VC). Increasing G_z from 0 to 1 and 1.8 G_z progressively shifted the volume-pressure curve of the chest wall to the left and also caused a fivefold exponential decrease in compliance. For lung volume <30% VC, gravity has an inspiratory effect, but this effect is much larger going from 0 to 1 G_z than from 1 to 1.8 G_z. For a volume from 30 to 70% VC, the effect is inspiratory going from 0 to 1 G_z but expiratory from 1 to 1.8 G_z. For a volume greater than ~70% VC, gravity always has an expiratory effect. The data suggest that the chest wall does not behave as a linear system when exposed to changing gravity, as the effect depends on both chest wall volume and magnitude of G_z.

The Combination of a Heat and Moisture Exchanger and a Booster™: A Clinical and Bacteriological Evaluation over 96 H—Thomachot L, Viviand X, Boyadjiev I, Vialet R, Martin C. Intensive Care Med 2002 Feb;28(2):147-153.

OBJECTIVE: To determine whether the combination with a new device (Booster™) for active humidification improves the efficacy of a hydrophobic heat and moisture exchanger (HME). DESIGN AND SETTING: Prospective, interventional study in the ICU of a university hospital. PATIENTS: Consecutive patients requiring controlled mechanical ventilation INTERVENTIONS: Patients were ventilated with a HME, and a Booster™ was added for 96 h to the ventilatory circuit. MEASUREMENTS AND RESULTS: During the inspiration phase the following factors were measured: peak and mean airway pressures, maximal (beginning of inspiration), minimal (end of inspiration), and mean values of temperature of inspired gases, and relative and absolute humidity of inspired gases. Microbiological samples were obtained from the Booster™, the ventilator side of the HME, and the tracheal secretions on days 1 and 4. Minimal and mean temperatures were increased as soon as the Booster™ was used and this increase was maintained for 96 h until the Booster™ was withdrawn. Then the temperature returned to baseline values. Absolute humidity values followed the same course. There was also some indirect evidence of very little, if any, changes in the HME resistance. The ventilatory side of the HMEs remained sterile in each patient, and the Booster™ was colonized by the same bacteria as those in the tracheal secretions. CONCLUSIONS: Adding the Booster™ to a hydrophobic HME improved the heat and water preservation of ventilatory gas.

Mortality Associated with Late-Onset Pneumonia in the Intensive Care Unit: Results of a Multi-Center Cohort Study—Moine P, Timsit JF, De Lassence A, Troche G, Fosse JP, Alberti C, Cohen Y. Intensive Care Med 2002 Feb;28(2):154-163.

OBJECTIVE: To evaluate the attributable mortality associated with late-onset nosocomial pneumonia (LOP) while taking into account the severity at admission, the evolution of the patients during the first 4 days after admission to the ICU and the appropriateness of initial empiric antibiotic treatment. DESIGN: Multicenter cohort study with prospective standardization of diagnostic interventions when nosocomial pneumonia develops. SETTING: Medical and surgical ICUs of four university-affiliated teaching hospitals. PATIENTS: Seven hundred sixty-four consecutive patients requiring ICU hospitalization for at least 4 days. MAIN OUTCOME MEASURES: The clinical and biological data as well as the therapeutic data and the outcome were prospectively recorded from the day of admission to ICU discharge. Simplified Acute Physiologic Score (SAPS II) and Logistic Organ Dysfunction (LOD) score were collected and computed within the first 4 calendar days of ICU admission. Variables associated with the outcome were selected using a stepwise Cox model. The time to acquisition of the first LOP was then introduced in the final model as a time-dependent covariate. The analysis was stratified by ICU center. Finally, as initial antibiotic therapy could have an impact on the increased risk of death induced by LOP, the Cox model was applied again introducing LOP immediately adequately treated and LOP not immediately adequately treated as two different time-dependent covariates. RESULTS: Late-onset pneumonia developed in 89 patients (12%). A McCabe score of more than 1, SAPS II score and increases in SAPS between days 1 and 2, days 2 and 3, and days 3 and 4 were significantly associated with an increased risk of death. When the time to acquisition

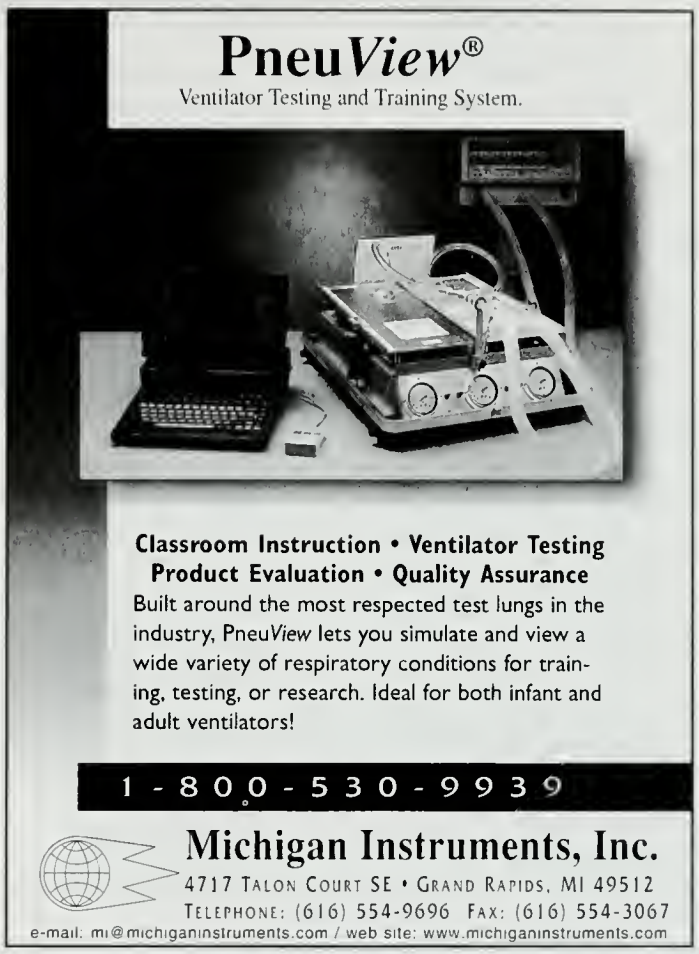
of the first episode of LOP was introduced into the Cox model, the LOP occurrence was associated with increased mortality, even adjusted over the selected prognostic parameters and after stratification by center (hazard ratio (HR)=1.53, 95% CI 1.02-2.3, p=0.04). When LOP immediately adequately treated and LOP not immediately adequately treated were separately introduced into the Cox model, inappropriately treated LOP remained significantly associated with an increased risk of mortality (HR=1.69, 95% CI 1.08-2.65, p=0.022), whereas appropriately treated LOP did not (HR=1.44, 95% CI 0.75-2.76, p=0.27). CONCLUSION: These data suggest that, in addition to severity scores, the underlying medical conditions and the evolution of severity within the first 4 days in ICU, late-onset pneumonia independently contribute to ICU patient mortality when empirical antibiotic treatment is not immediately appropriate.

Physiological Effects of Constant Versus Decelerating Inflation Flow in Patients with Chronic Obstructive Pulmonary Disease Under Controlled Mechanical Ventilation—Guerin C, Lemasson S, La Cara F, Fournier G. Intensive Care Med 2002 Feb;28(2):164-169.

OBJECTIVE: To study the cardiorespiratory effects of inspiratory flow rate and waveform in COPD patients. DESIGN: Prospective physiological investigation with randomized allocations of experimental conditions. SETTING: A 14-bed medical ICU in a 1000-bed university hospital. PATIENTS AND PARTICIPANTS: Ten COPD intubated, sedated and paralyzed patients with chronic obstructive pulmonary disease (COPD), mechanically ventilated for acute respiratory failure. INTERVENTIONS: In volume-controlled mode, three inflation flow rates of 0.40, 0.70, and 1.10 L/s for 20 min with a constant (CF) or a decelerating (DF) inflation flow profile. Each patient received all six experimental conditions in a random order. Tidal volume and respiratory frequency were similar during the experimental conditions. MEASUREMENTS AND RESULTS: Arterial blood gases, hemodynamics (n=8), and respiratory mechanics were measured with zero end expiratory pressure. Between flow rates the median (25th-75th percentiles) values of P_{aO_2}/F_{IO_2} were 232 (132-289), 253 (161-338), 231 (163-352) for CF and 253 (143-331), 249 (164-360), 231 mm Hg (187-351), for DF, respectively; the maximal airway pressures were 25.6, 28.3, 34.6 cm H_2O for CF and 21.7, 29.6, 34.8 cm H_2O for DF, respectively, the mean airway pressures were 8.9, 6.1, 5.4 cm H_2O for CF and 9.1, 7, 6.5 cm H_2O for DF, respectively. CONCLUSIONS: Changing the ventilator in volume-controlled mode with a DF or CF profile has no significant cardiorespiratory effect in intubated COPD patients mechanically ventilated for acute respiratory failure.

Cognitive and Perceived Health Status in Patient with Chronic Obstructive Pulmonary Disease Surviving Acute on Chronic Respiratory Failure: A Controlled Study—Ambrosino N, Bruletti G, Scala V, Porta R, Vitacca M. Intensive Care Med 2002 Feb;28(2):170-177.

OBJECTIVE: To evaluate the perceived health and cognitive status in survivors of chronic obstructive pulmonary disease (COPD) exacerbations requiring mechanical ventilation. DESIGN AND SETTING: Prospective controlled cohort study in a respiratory intermediate intensive care unit. PATIENTS: Sixty-three COPD patients at their first episode of acute on chronic respiratory failure requiring mechanical ventilation, controls were 34 consecutive stable COPD patients on long-term oxygen therapy with no previous ICU admission. MEASUREMENTS AND RESULTS: Perceived health and cognitive status by means of the Nottingham Health Profile (NHP) and the Mini Mental State (MMS), respectively, at discharge and 3 and 6 months thereafter. At discharge patients showed significantly worse mean values than controls in MMS and in all NHP dimensions except pain. The MMS score was below 24, the threshold level of cognitive impairment, in 43% patients of the study group but only 3% of controls (p=0.006). Six months after discharge patients showed NHP and MMS values similar to those of controls and the proportion of patients with a MMS score below 24 had significantly decreased to 17%, a value not significantly different from that in controls (5%). CONCLUSIONS: COPD patients surviving their first episode of acute on chronic respiratory failure requiring mechanical ventilation, suffer worsen perceived health status and cognitive function, which 6 months after discharge may improve to levels similar to those in stable COPD patients on long-term oxygen therapy with no previous ICU admission.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Is the Bispectral Index Appropriate for Monitoring the Sedation Level of Mechanically Ventilated Surgical ICU Patients?—Frenzel D, Greim CA, Sommer C, Bauerle K, Roewer N. Intensive Care Med 2002 Feb;28(2):178-183.

OBJECTIVE: To determine the value of the bispectral index (BIS) in assessing the depth of sedation in sedated and mechanically ventilated ICU patients, compared with clinical sedation scores. DESIGN AND SETTING: Prospective convenience sample in a 12-bed anesthesiological-surgical ICU of a university hospital. PATIENTS: 19 consecutive patients without any central neurological diseases requiring mechanical ventilation for more than 24 h. MEASUREMENTS: BIS version 3.12 and clinical depth of sedation assessed by the modified Observers's Assessment of Alertness/Sedation Scale, modified Glasgow Coma Scale, modified Ramsay Scale, Cook Scale, and Sedation-Agitation Scale were measured twice daily while patients were intubated and once daily after extubation until discharged from ICU. RESULTS: there was a moderate correlation between BIS and each sedation score in 11 patients (58%, "BIS patients") and no correlation in 8 patients (42%, "non-BIS

patients"). We found no parameters distinguishing between these two groups. On average eight measurements were necessary to establish a statistical correlation. In the BIS patients the slopes of the linear regression curves showed significant differences for all BIS score combinations with increasing scattering at deeper sedation levels. CONCLUSIONS: BIS is correlated only in some ICU patients with the clinical assessment of their sedation level as based on various scores. At deeper sedation levels the interindividual differences increase. There were no criteria found to distinguish patients with and without correlation. This suggests that the BIS is not suitable for monitoring the sedation in a heterogeneous group of surgical ICU patients.

Epidemiology, Etiology, and Prevention of Lung Cancer—Bilello KS, Murin S, Matthay RA. Clin Chest Med 2002 Mar;23(1):1-25.

Over the past century, lung cancer has gone from an obscure disease to the leading cause of cancer death worldwide. Initially an epidemic disease among men in industrialized nations, lung cancer now has become the leading cancer killer in both sexes in the United States and an increasingly common disease of both sexes in developing countries. Lung cancer incidence largely mirrors smoking prevalence, with a latency period of several decades. Other important risk factors for the development of lung cancer include environmental exposure to tobacco smoke, radon, occupational carcinogens, and pre-existing nonmalignant lung disease. Studies in molecular biology have elucidated the role that genetic factors play in modifying an individual's risk for lung cancer. Although chemopreventive agents may be developed to prevent lung cancer, prevention of smoking initiation and promotion of smoking cessation are currently the best weapons to fight lung cancer. No other malignancy has been shown to have such a strong epidemiologic relation between a preventable behavior and incidence of disease. Despite this knowledge, more than 20% of all Americans smoke, and tobacco use is exploding in developing countries. Based on current and projected smoking patterns, it is anticipated that lung cancer will remain the leading cause of cancer death in the world for decades to come.

Preventing Lung Cancer by Stopping Smoking—Hurt RD, Ebbert JO. Clin Chest Med 2002 Mar;23(1):27-36, vii.

Much progress has been made in recent years in treating tobacco dependence. The 2000 USPHS Guideline extends the understanding of effective treatments and encourages clinicians to be more diligent in recognizing tobacco users in the practice and more aggressive in treating every tobacco user. The guideline outlines the potential use of the five first-line medications (bupropion, nicotine patches, gum, nasal spray, and inhaler) and the two second-line medications (nortriptyline and clonidine). The use of these medications can be tailored to meet the patients needs and combinations of these medications can be used when appropriate. More intensive treatment, such as residential treatment, may be needed for more addicted smokers.

Bronchoscopy and Needle Biopsy Techniques for Diagnosis and Staging of Lung Cancer—Mazzone P, Jain P, Arroliga AC, Matthay RA. Clin Chest Med 2002 Mar;23(1):137-158, ix.

Lung cancer is the leading cause of cancer deaths in the United States. The individual therapeutic approach and prognosis depends on accurate diagnosis and staging. Flexible bronchoscopy (FB) and transthoracic needle biopsy (TNB) are the most widely used techniques for this purpose. This article provides a critical overview of indications, diagnostic yield, and limitations of bronchoscopy and TNB in the diagnosis of lung cancer.

Functional Evaluation Before Lung Resection—Schuurmans MM, Diacon AH, Bolliger CT. Clin Chest Med 2002 Mar;23(1):159-172.

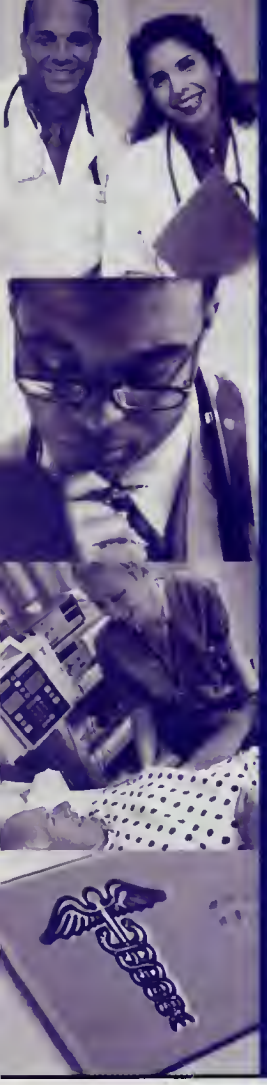
Advances in operative technique and perioperative care have reduced surgical morbidity and mortality considerably after pulmonary resections. Various single and combined parameters of functional operability have been proposed to assess the surgical risk. Patients with normal or only slightly impaired pulmonary function (FEV_1 and DLCO $\geq$ 80% predicted) and no cardiovascular risk factors can undergo pulmonary resections up to a pneumonectomy without further investigation. For others, exercise testing, pulmonary split-function studies, or a combination of these methods are recommended. Cardiopulmonary exercise testing, most frequently performed as a symptom-limited test with the measurement of $\dot{V}_{O_2max}$, assesses the pulmonary and cardiovascular reserves. A $\dot{V}_{O_2max}$ of < 10 mL/kg/minute generally is considered prohibitive for any resection, a value of > 20 mL/kg/minute or greater than 75% predicted normal, safe for major resections. Split-function studies are radionuclide-based estimations of the ppo values of various parameters. The currently used ppo parameters are FEV_1-ppo, DLCO-ppo, and $\dot{V}_{O_2max}$ -ppo. Suggested cutoff values for safe resection are: FEV_1-ppo and DLCO-ppo 40% or > predicted, and $\dot{V}^rO_{2max\text{-}ppo}$ 35% or greater than predicted, combined with an absolute value of greater than or equal to 10 mL/kg/minute. The lowest acceptable ppo values remain to be confirmed by additional prospective studies. Resections involving not more than one lobe usually lead to an early functional deficit followed by recovery. The permanent loss in pulmonary function is small (approximately 10%) and exercise capacity is reduced only slightly or not at all. Pneumonectomy leads to an early permanent loss of about 33% in pulmonary function and approximately 20% in exercise capacity. Pulmonary function tests alone therefore overestimate the functional loss after lung resection.

Therapeutic Bronchoscopy in Lung Cancer: Laser Therapy, Electrocautery, Brachytherapy, Stents, and Photodynamic Therapy—Lee P, Kupeli E, Mehta AC. Clin Chest Med 2002 Mar;23(1):241-256.

Therapeutic bronchoscopic techniques such as LPR, EC, brachytherapy, stents, and PDT are effective tools in the palliation and local control of lung cancer. Palliation of malignant tracheobronchial obstruction by LPR, stents, brachytherapy, PDT, or a combination thereof results in relief of dyspnea, hemoptysis, and postobstructive pneumonia. Importantly, it avoids intubation in patients with respiratory distress and facilitates the weaning of patients from MV. In the exciting field of lung cancer screening and treatment of early lung cancer, PDT, brachytherapy, EC, and LPR may represent treatment alternatives to surgical resection, especially in a select group of patients with high surgical risk or favorable endobronchial lesions. Clinicians await the results of future studies, which will (1) better define the impact of each treatment modality on patient care in terms of cost, survival, and improvement in quality of life, and (2) determine the optimal combination therapy relative to bronchoscopic and conventional treatment for effective palliation and cure of lung cancer.

Predicting Pulmonary Complications After Nonthoracic Surgery: A Systematic Review of Blinded Studies—Fisher BW, Majumdar SR, McAlister FA. Am J Med 2002 Feb 15;112(3):219-225.

PURPOSE: To determine the performance of variables commonly used in the prediction of postoperative pulmonary complications in patients undergoing nonthoracic surgery. METHODS: We conducted a systematic review of the literature in English, using MEDLINE (1966-2001), manual searches of identified articles, and contact with content experts. All studies reporting independent and blinded comparisons of preoperative or operative factors with postoperative pulmonary complications were included. Two reviewers independently abstracted inclusion and exclusion criteria, study designs, patient characteristics, predictors of interest, and the nature and occurrence of postoperative pulmonary complications. RESULTS: Seven studies fulfilled the inclusion criteria. The definition of postoperative pulmonary complications differed among



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studies, and the incidence of postoperative pulmonary complications varied from 2% to 19%. Of the 28 preoperative or operative predictors that were evaluated in the 7 studies, 16 were associated significantly with postoperative pulmonary complications, although only 2 (duration of anesthesia and postoperative nasogastric tube placement) were significant in more than one study. The positive (2.2 to 5.1) and negative (0.2 to 0.8) likelihood ratios for these 16 variables suggest that they have only modest predictive value. Neither hypercarbia nor reduced spirometry values were independently associated with an increased risk of postoperative pulmonary complications. CONCLUSION: Few studies have rigorously evaluated the performance of the preoperative or operative variables in the prediction of postoperative pulmonary complications. Prospective studies with independent and blinded comparisons of these variables with postoperative outcomes are needed.

Noninvasive Continuous Monitoring of the Effects of Head Position on Brain Hemodynamics in Ventilated Infants—Pellicer A, Gaya F, Madero R, Quero J, Cabanas F. Pediatrics 2002 Mar;109(3):434-440.

HYPOTHESIS: Laying supine with the head in midline position improves cerebral venous return by preventing functional occlusion of the vessels of the neck. OBJECTIVES: To assess changes in cerebral blood volume (ΔCBV) and cerebral blood flow (CBF) with the position of the head in ventilated patients using a noninvasive method. The influence of the type of ventilation and birth weight was evaluated. METHODS: Thirteen conventionally ventilated and 8 high-frequency oscillatory ventilated infants, with mean gestational ages and birth weights of 31 ± 5 weeks (24–38) and 1575 ± 803 g (560–3000), respectively, were studied 5.8 ± 7.8 days (1–33) after birth. ΔCBV (mL/100 g) and CBF (mL/100 g/min) were measured by near-infrared spectroscopy with the head in supine midline position (ΔCBV_s, CBF_s) and rotated 90 to one side (ΔCBV_{lat}, CBF_{lat}). Heart rate, peripheral saturation, transcutaneous P_{CO_2}, and blood pressure were monitored continuously. Ventilatory settings remained constant throughout the study period. RESULTS: Mean ΔCBV_s was lower than mean ΔCBV_{lat}, although no changes in blood pressure, transcutaneous P_{CO_2}, oxygenation, or heart rate occurred. This change in ΔCBV was not associated with the type of ventilation or birth weight, but the differences tended to be greater ($d\Delta CBV = \Delta CBV_{lat} - \Delta CBV_s$) in the smallest infants (<1200 g). In contrast, CBF did not vary. CONCLUSION: The supine midline position of the head favors cerebral venous drainage and helps to prevent elevation of CBV. SPECULATION: This finding may be important in the first days of life, particularly in tiny preterm infants recovering from lung disease with improving lung compliance, in which functional obstruction of cerebral venous drainage should be avoided.

Inattention, Hyperactivity, and Symptoms of Sleep-Disordered Breathing—Chervin RD, Archbold KH, Dillon JE, Panahi P, Pituch KJ, Dahl RE, Guilleminault C. Pediatrics 2002 Mar;109(3):449-456.

OBJECTIVE: Inattention and hyperactivity are frequent among children with sleep-disordered breathing (SDB) and often improve when SDB is treated. However, the frequency of SDB symptoms among inattentive and hyperactive children has received little study. DESIGN: Cross-sectional survey. SETTING: Two university-affiliated but community-based general pediatrics clinics. PATIENTS: Patients consisted of N = 866 children (469 boys), aged 2.0 to 13.9 years (mean: 6.8 ± 3.2 years), with clinic appointments. MEASURES: A validated Pediatric Sleep Questionnaire assessed for habitual snoring (1 item), snoring severity (a 4-item subscale), sleepiness (4 items), and overall risk of SDB (16 items). Parents also completed 2 common behavioral measures, an inattention/hyperactivity scale (IHS) derived from the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, and the hyperactivity index (HI, expressed as a t score) of the Conners' Parent Rating Scale. RESULTS: Habitual snoring was reported in 16% (95% confidence interval [CI]: 13, 19) of the participants. High HI scores (>60) were found in 13% (95% CI: 11, 16) of all participants, 22% (95% CI: 15, 29) of habitual snorers, and 12% (95% CI: 9, 14) of nonsnorers. Odds ratios between HI >60 and each of the following were: habitual snoring, 2.2 (95% CI: 1.4, 3.6); 1 additional positive symptom-item on the snoring scale, 1.3 (95% CI: 1.1, 1.5); 1 additional positive item on the sleepiness scale, 1.6 (95% CI: 1.4, 2.0); and a 1-standard deviation increase in the overall SDB score, 1.7 (95% CI: 1.4, 2.0; all odds ratios age- and sex-adjusted). Results were similar for high IHS scores (>1.25). Stratification by age and sex showed that most of the association with snoring (but not sleepiness) derived from boys <8 years old. CONCLUSIONS: Inattention and hyperactivity among general pediatric patients are associated with increased daytime sleepiness and—especially in young boys—snoring and other symptoms of SDB. If sleepiness and SDB do influence daytime behavior, the current results suggest a major public health impact.

Practice Variation in the Emergency Management of Croup—Hampers LC, Faries SG. Pediatrics 2002 Mar;109(3):505-508.

OBJECTIVE: To explore the effect of physician training background on the emergency management of croup. METHODS: Two community hospitals with a general emergency department (ED) staffed by board-certified emergency medicine (EM) practitioners were the setting for this study. At both sites, pediatricians (PED) or pediatric emergency medicine specialists (PEM) managed acute pediatric visits during evening and weekend hours. Retrospective patient cohorts (6 months to 6 years) with a primary discharge diagnosis of croup were identified from a 1-year period. Data abstraction was performed by a registered nurse who was blinded to the study hypothesis. RESULTS: There were 229, 92, and 209 patients in the PED, PEM, and EM cohorts, respectively, reflecting the practice of 69 physicians (19 PED, 12 PEM, and 38 EM). The groups had similar rates of admission and prescription of steroids at discharge. In regression models that incorporated all recorded clinical variables, EM patients were more likely to have received a chest radiograph (odds ratio [OR]: 6.6; 95% confidence interval [CI]: 3.1–14), racemic epinephrine (OR: 6.5; 95% CI: 3.1–14), albuterol in the ED (OR: 3.0; 95% CI: 1.4–6.4), and parenteral steroids (OR: 3.6; 95% CI: 2.1–6.3) and were less likely to have received oral steroids (OR: 0.41; 95% CI: 0.26–0.64). For the EM cohort, adjusted mean length of ED visit was 40 minutes longer (95% CI: 6.8–72) and mean direct costs were $90 higher (95% CI: $27–$153). Regression models comparing the PEM and PED cohorts revealed no significant management differences. CONCLUSION: Compared with physicians with a pediatric background, rates of resource utilization were higher for EM-trained physicians who managed uncomplicated cases of croup.

Pulmonary Function in Technology-Dependent Children 2 Years and Older with Bronchopulmonary Dysplasia—Talmaciu I, Ren CL, Kolb SM, Hickey E, Panitch HB. Pediatr Pulmonol 2002 Mar;33(3):181-188.

Somatic and pulmonary growth coincide with resolution of hypoxemia by 2 years of age in most children with bronchopulmonary dysplasia (BPD). However, a distinct subgroup of children with BPD continue to require mechanical ventilation and/or supplemental oxygen beyond 2 years of age. This study tested the hypothesis that indices of pulmonary function would be significantly worse in children with BPD 2 years and older who remained technology-dependent secondary to hypoxemia, compared to those of age-matched children with BPD who were normoxemic. We measured pulmonary mechanics in 21 oxygen- or ventilator-dependent children with BPD 2 years and older ($BPDO_2$ group; mean age±SD, 30.2±6.5 months) and in 19 children with BPD who had been weaned off mechanical ventilation and supplemental oxygen for at least 6 months (control group; mean age, 30.1±5.5 months). Respiratory rate and tidal volume were measured after sedation with chloral hydrate, and dynamic

compliance and expiratory conductance were calculated using the esophageal catheter technique. Maximal flow at FRC ($\dot{V}_{maxFRC}$) and ratio of forced-to-tidal flows at midtidal volume were obtained by the rapid thoracic compression technique. FRC was determined by nitrogen washout. There were no statistically significant differences in most measured indices of pulmonary mechanics between the BPDO2 and control groups. However, $\dot{V}_{maxFRC}$/FRC was higher in controls compared to subjects in the BPDO2 group (0.81±0.40 sec^{-1} vs. 0.34±0.21 sec^{-1}, $p<0.003$). We conclude that most indices of pulmonary function in children with BPD 2 years and older do not reflect the need for mechanical ventilation or supplemental oxygen. We speculate that measurements of lung elastic recoil and tests of distribution of ventilation and pulmonary perfusion may be more sensitive in differentiating normoxemic and hypoxemic children with BPD 2 years and older.

Salbutamol Dry Powder Inhaler: Efficacy, Tolerability, and Acceptability Study—O'Callaghan C, Everard ML, Bush A, Hiller EJ, Ross-Russell R, O'Keefe P, Weller P. Pediatr Pulmonol 2002 Mar;33(3):189-193.

Dry powder inhaler (DPI) devices are frequently used in children over 5 years of age in order to avoid coordination difficulties often seen with the use of pressurized metered dose inhalers (pMDI). This study assessed the efficacy, tolerability, and acceptability of salbutamol delivered via two delivery systems, in a population of pediatric patients. The primary aim of the study was to investigate the bronchodilator efficacy of a single dose (100 microg) of salbutamol administered via a dry powder inhaler (Clickhaler) compared to a similar dose administered by a pressurized metered dose inhaler via a large-valved holding chamber (VHC) to children with asthma. The study comprised two phases: the first comparator phase, followed by an open 4-week treatment period. Sixty-one children with a mean (SD) age of 11.3 years (2.9) (range, 6-17) and mild or moderate asthma completed the study. The primary efficacy endpoint, forced expiratory volume in 1 sec (FEV_1), indicated that there was no clinically or statistically significant difference between the bronchodilator effects of salbutamol delivered via either device, with a maximum posttreatment percentage change in FEV_1 (SD) of 12.4% (10.0) and 14.15 (9.3) for Clickhaler and pMDI plus VHC, respectively. Most patients rated the Clickhaler as easy to use (97%) and liked the device (84%). Both treatments were well-tolerated. These results support the suitability of salbutamol Clickhaler as an acceptable, well-tolerated, and effective alternative to a pMDI plus VHC in mild to moderate asthmatic children over age 6 years.

Randomized Controlled Study of In-Hospital Exercise Training Programs in Children with Cystic Fibrosis—Selvadurai HC, Blimkie CJ, Meyers N, Mellis CM, Cooper PJ, Van Asperen PP. Pediatr Pulmonol 2002 Mar;33(3):194-200.

The aim of this study was to compare aerobic and resistance training in children with cystic fibrosis (CF) admitted to hospital with an intercurrent pulmonary infection with a control group. The subjects were randomized into three groups on the first day of admission. The fat-free mass (FFM) was calculated, using the skin fold thickness from four sites (biceps, triceps, subscapular, and iliac crest). Pulmonary function tests were performed within 36 hr of admission and repeated on discharge from the hospital, and again at 1 month after discharge. All subjects performed an incremental treadmill exercise test, using a modified Bruce protocol. Lower limb strength was measured using a Cybex dynamometer. An assessment of quality of life was made using the Quality of Well Being Scale, as previously reported. Activity levels were measured using a 7-day activity diary, and subjects also wore an accelerometer on their hips. There were no significant differences between the three groups in terms of disease severity, and length of stay in hospital. Subjects in all three groups received intravenous antibiotics and nutritional supplementation as determined by the physician. Children randomized to the aerobic training group participated in aerobic activities for five sessions, each of 30-min duration, a week. The children randomized to the resistance training group exercised both upper and lower limbs against a graded resistance machine. Subjects in the control group received standard chest physiotherapy. Our study demonstrated that children who received aerobic training had significantly better peak aerobic capacity, activity levels, and quality of life than children who received the resistance training program. Children who received resistance training had better weight gain (total mass, as well as fat-free mass), lung function, and leg strength than children who received aerobic training. A combination of aerobic and resistance training may be the best training program, and future studies to assess optimal training programs for CF patients are indicated.

Safety of Inhaled Corticosteroids in Children—Allen DB. Pediatr Pulmonol 2002 Mar;33(3):208-220.

Inhaled corticosteroids (ICS) are now first-line therapy for persistent asthma in children. The major safety concerns of long-term ICS therapy for childhood asthma are potential effects on adrenal function, growth, and bone mass. Dosage, type of inhaler device, and individual drug characteristics influence systemic effects of ICS. Sensitive measures of basal adrenal function can show statistically significant changes during ICS therapy, but these do not accurately predict clinically meaningful adrenal axis suppression. Adrenal insufficiency is rare and confined to children receiving high doses of ICS. Dose-related inhibition of growth has been seen in some short- and intermediate-term studies, but long-term studies have found no detrimental effect on final height. ICS therapy has not been

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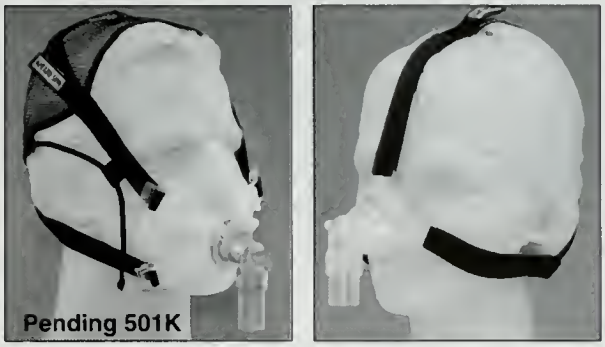

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associated with significant changes in measurements of bone and bone biomarkers, but more studies of high doses and of therapy in adolescents are needed. Overall, although ICS are the most effective anti-inflammatory treatment available for asthma, high doses of ICS in children are still of concern. The risk of high doses is compounded in children with concomitant allergic conditions that require multiple forms of topical corticosteroids. Benefits of ICS clearly outweigh potential adverse effects and risks associated with poorly controlled asthma. Risk can be minimized by using the lowest effective ICS dose, limiting systemic availability of the drug through proper technique to minimize swallowed drug, and selection of agents with efficient first-path hepatic inactivation of swallowed drug. Adjuvant treatments can reduce the dose of ICS required for asthma control, allowing a reduction in overall systemic exposure for most children with mild-to-moderate persistent asthma. Therefore, these agents should be added to, but should not replace, ICS therapy.

Laryngeal Hirudiniasis: An Unusual Cause of Airway Obstruction and Hemoptysis—Mohammad Y, Rostum M, Dubaybo BA. Pediatr Pulmonol 2002 Mar;33(3):224-226.

Cases of childhood hemoptysis are rare and usually result from foreign body aspiration or congenital heart or lung diseases. However, human hirudiniasis due to the leech still exists, and could involve the upper airways after drinking infested water from quiet streams and pools. We report the case of a 6-year-old child who presented suffocating at the emergency room after having been misdiagnosed and treated for asthma over a 1-month period. His mother reported he had had recurrent hemoptysis, as well. The child inadvertently drank leech-infested water in a rural area of northern Syria. Surgical removal of the leech resulted in prompt resolution of the symptoms. Although laryngeal hirudiniasis is rare in the developed world, it remains a possible cause of childhood airway obstruction, hemoptysis, and anemia which needs to be considered in patients with a suggestive history.

Selective Fiberoptic Left Main-Stem Intubation for Severe Unilateral Barotrauma in a 24-Week Premature Infant—Meyer MT, Rice TB, Glaspey JC. Pediatr Pulmonol 2002 Mar;33(3):227-231.

A 24-week premature infant developed severe right-sided pulmonary barotrauma secondary to mechanical ventilation for respiratory distress syndrome (RDS). High-frequency oscillatory ventilation and permissive hypercapnia were initiated. A chest tube was placed to relieve a pneumothorax, and a catheter was inserted into an air-filled cyst for drainage. These maneuvers failed to improve the child's respiratory status. The child's left main-stem bronchus was then successfully fiberoptically intubated for single-lung ventilation in order to reduce the unilateral barotrauma. Single-lung ventilation was effectively and safely continued for 5 days, with complete resolution of the pulmonary barotrauma.

Frequency, Severity, and Duration of Rhinovirus Infections in Asthmatic and Non-Asthmatic Individuals: A Longitudinal Cohort Study—Corne JM, Marshall C, Smith S, Schreiber J, Sanderson G, Holgate ST, Johnston SL. Lancet 2002 Mar 9;359(9309):831-834.

BACKGROUND: Rhinovirus infections cause exacerbations of asthma. We postulated that people with asthma are more susceptible to rhinovirus infection than people without the disease and compared the susceptibility of these groups. METHODS: We recruited 76 cohabiting couples. One person in every couple had atopic asthma and one was healthy. Participants completed daily diary cards of upper-respiratory-tract (URT) and lower-respiratory-tract (LRT) symptoms and measured peak expiratory flow twice daily. Every 2 weeks nasal aspirates were taken and examined for rhinovirus. Mixed models were used to compare risks of infection between groups. We also compared the severity and duration of infections. FINDINGS: We analysed 753 samples. Rhinovirus was detected in 10.1% (38/378) of samples from participants with asthma and 8.5% (32/375) of samples from healthy participants. After adjustment for confounding factors, asthma did not significantly increase risk of infection (odds ratio 1.15, 95% CI 0.71-1.87). Groups did not differ in frequency, severity, or duration of URT infections or symptoms associated with rhinovirus infection. First rhinovirus infection was associated more frequently with LRT infection in participants with asthma than in healthy individuals (12 of 28 infections vs four of 23, respectively, p=0.051). Symptoms of LRT associated with rhinovirus infection were significantly more severe (p=0.001) and longer-lasting in participants with asthma than in healthy participants (p=0.005). INTERPRETATION: People with atopic asthma are not at greater risk of rhinovirus infection than healthy individuals but suffer from more frequent LRT infections and have more severe and longer-lasting LRT symptoms.

The Effect of Chin Lift, Jaw Thrust, and Continuous Positive Airway Pressure on the Size of the Glottic Opening and on Stridor Score in Anesthetized, Spontaneously Breathing Children—Meier S, Geiduschek J, Paganoni R, Fuehrmeyer F, Reber A. Anesth Analg 2002 Mar;94(3):494-499.

Chin lift and jaw thrust are two common maneuvers used to improve the patency of the upper airway during general anesthesia. We investigated the effect of these maneuvers combined with continuous positive airway pressure (CPAP) on the size of glottic opening and on stridor score. Forty children, aged 2–9 yr, premedicated with midazolam and spontaneously breathing end-tidal 1% halothane and equal parts of nitrous oxide and oxygen, were studied. A flexible fiberoptic bronchoscope was placed via mask and one nostril to the level of the junction of the soft palate and oropharynx. Video recordings and simultaneous stridor scores were obtained during six conditions: 1) chin unsupported, 2) manual chin lift, 3) chin lift and CPAP 10 cm H_2O, 4) repeat chin unsupported, 5) manual jaw thrust, and 6) jaw thrust and CPAP 10 cm H_2O. Videos were analyzed to determine the percentage of glottic opening (POGO) score. POGO score increased ($p < 0.05$) in Conditions 2, 3, 5, and 6. With increasing POGO score there was a decrease in stridor score ($p < 0.05$). IMPLICATIONS: Chin lift and jaw thrust maneuvers combined with continuous positive airway pressure improve the view of the glottic opening as viewed by flexible nasal laryngoscopy and decrease stridor in anesthetized, spontaneously breathing children.

The Correlation of the Bispectral Index Monitor with Clinical Sedation Scores During Mechanical Ventilation in the Pediatric Intensive Care Unit—Berkenbosch JW, Fichter CR, Tobias JD. Anesth Analg 2002 Mar;94(3):506-511.

In patients who are mechanically ventilated in the pediatric intensive care unit (PICU), sedative and/or analgesic medications are routinely provided and titrated to effect based on clinical assessment of the patient. The bispectral index (BIS) monitor uses a modified electroencephalogram to quantify the effects of central nervous system-acting drugs on the level of consciousness. To evaluate the usefulness of the BIS monitor to predict clinical sedation levels in the PICU, we compared BIS values with simultaneously obtained clinical sedation scores in 24 mechanically ventilated pediatric patients aged 5.7 ± 6.1 yr. For each sedation scale used, the BIS value correlated with increasing depth of sedation ($p < 0.0001$) and was independent of the drug(s) used for sedation. To differentiate adequate from inadequate sedation, a BIS value <70 had a sensitivity of 0.87–0.89 and a positive predictive value of 0.68–0.84. To differentiate adequate from excessive sedation, a BIS value <50 had a sensitivity of 0.67–0.75 and a positive predictive value of 0.07–0.52. We conclude that the BIS monitor may be a useful adjunct for the assessment of sedation in PICU patients. IMPLICATIONS: We demonstrate the usefulness of the bispectral index monitor for assessing sedation in pediatric intensive care unit patients. The bispectral index monitor correlated with clinically assessed

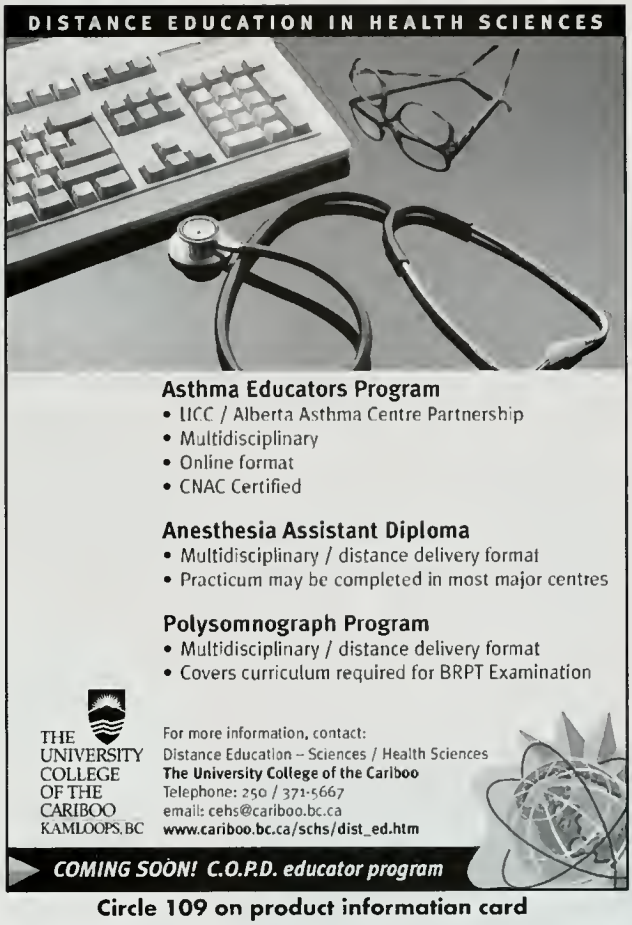
sedation levels and was useful for differentiating adequate from inadequate sedation, which would be of value when the clinical examination is unavailable.

Morbid Obesity and Tracheal Intubation—Brodsky JB, Lemmens HJ, Brock-Utne JG, Vierra M, Saidman LJ. Anesth Analg 2002 Mar;94(3):732-736.

The tracheas of obese patients may be more difficult to intubate than those of normal-weight patients. We studied 100 morbidly obese patients (body mass index >40 kg/m^2) to identify which factors complicate direct laryngoscopy and tracheal intubation. Preoperative measurements (height, weight, neck circumference, width of mouth opening, sternomental distance, and thyromental distance) and Mallampati score were recorded. The view during direct laryngoscopy was graded, and the number of attempts at tracheal intubation was recorded. Neither absolute obesity nor body mass index was associated with intubation difficulties. Large neck circumference and high Mallampati score were the only predictors of potential intubation problems. Because in all but one patient the trachea was intubated successfully by direct laryngoscopy, the neck circumference that requires an intervention such as fiberoptic bronchoscopy to establish an airway remains unknown. We conclude that obesity alone is not predictive of tracheal intubation difficulties. IMPLICATIONS: In 100 morbidly obese patients, neither obesity nor body mass index predicted problems with tracheal intubation. However, a high Mallampati score (≤ 3) and large neck circumference may increase the potential for difficult laryngoscopy and intubation.

The Laryngeal Mask Airway ProSeal™ As a Temporary Ventilatory Device in Grossly and Morbidly Obese Patients Before Laryngoscope-Guided Tracheal Intubation—Keller C, Brimacombe J, Kleinsasser A, Brimacombe L. Anesth Analg 2002 Mar;94(3):737-740.

We determined the efficacy of the laryngeal mask airway ProSeal™ (PLMA) as a temporary ventilatory device in morbidly obese patients before laryngoscope-guided tracheal intubation. Sixty patients (body mass index 35–60 kg/m^2) scheduled for elective surgery, who preferred airway management under general anesthesia, were studied. The induction of anesthesia was with midazolam/fentanyl/propofol and maintenance was with sevoflurane 1%–3% in oxygen 100%. The PLMA was inserted and an effective airway established. Rocuronium was given I.V. for paralysis. Oropharyngeal leak pressure, ease of gastric tube placement, residual gastric volume, fiberoptic position of the airway/drainage tube, and ease of ventilation at a tidal volume of 8 mL/kg was determined. The PLMA was then removed and laryngoscope-guided tracheal intubation attempted. The number of insertion/intubation attempts (maximum two each) and time taken to establish an effective airway with each device were recorded. An effective airway was obtained at the first insertion attempt in 90% of patients (54/60) and at the second attempt in 10% (6/60). The time taken to provide an effective airway was 15 ± 7 s (7–42 s). Oropharyngeal leak pressure was 32 ± 8 cm H_2O (12—40 cm H_2O. The residual gastric volume was 36 ± 46 mL (0–240 mL). Positive pressure ventilation without air leaks was possible in 95% of patients (57/60). The vocal cords were seen from the airway tube in 75% of patients (45/60), but the esophagus was not seen. The fiberoptic view from the drainage tube revealed mucosa in 93% of patients (56/60) and an open upper esophageal sphincter in 7% (4/60). Tracheal intubation was successful at the first attempt in 90% of patients (54/60), at the second attempt in 7% (4/60), and failed in 3% (2/60). In these latter two patients, the PLMA was reinserted and surgery performed uneventfully with the PLMA. The time taken to tracheally intubate the patient was 13 ± 10 s (8–51 s). There were no episodes of hypoxia (S_{PO_2} <90%) or other adverse events. There were no differences in insertion success rate, or the time to successful insertion between the PLMA and laryngoscope-guided intubation. We conclude that the PLMA is an effective temporary ventilatory device in grossly or morbidly obese patients before laryngoscope-guided tracheal intubation. IMPLICATIONS: The laryngeal mask airway ProSeal™ is an effective temporary ventilatory device in grossly and morbidly obese patients before laryngoscope-guided tracheal intubation.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Internet-Based Home Monitoring of Pulmonary Function After Lung Transplantation—Morlion B, Knoop C, Paiva M, Estenne M. Am J Respir Crit Care Med 2002 Mar 1;165(5):694-697.

Home monitoring of spirometry has been advocated in lung transplant recipients for the early detection of acute infection and rejection of the allograft. We have developed a user-friendly, Internet-based telemonitoring system providing direct transmission of home spirometry to the hospital. In this prospective study, we assessed patient adherence with the monitoring, agreement between home and hospital spirometry, intrasubject coefficient of variation (CV) for FEV_1 and FEF_{25-75}, and sensitivity of these variables for the detection of acute complications. Twenty-two bilateral-lung and heart-lung transplant recipients were followed for a median of 473 d (range, 60-822), during which 13,833 measurements were obtained. Patient compliance was 55% for two measurements a day and 84% for one measurement a day. Agreement between home and hospital spirometry was within 4% for FEV_1 and 6% for FEF_{25-75}. Mean CV was 3.2% for FEV_1 and 7.5% for FEF_{25-75}. Using transbronchial lung biopsy and/or bronchoalveolar lavage as gold standards, the sensitivity of home spirometry was 63%, and 23% of true positives were detected by changes in FEF_{25-75} alone. We conclude that home monitoring of pulmonary function in lung transplant recipients via the Internet is feasible

and provides very reproducible data; yet it has only a mild sensitivity for the detection of acute allograft dysfunction.

Comparison of Nebulized Budesonide and Oral Prednisolone with Placebo in the Treatment of Acute Exacerbations of Chronic Obstructive Pulmonary Disease: A Randomized Controlled Trial—Maltais F, Ostinelli J, Bourbeau J, Tonnel AB, Jacquemet N, Haddon J, et al. Am J Respir Crit Care Med 2002 Mar 1;165(5):698-703.

Nebulized budesonide has been used successfully to treat acute asthma exacerbation, and we hypothesized that it could also be effective for exacerbations of chronic obstructive pulmonary disease (COPD). In this multicenter, double-blind, randomized, placebo-controlled trial, the efficacy of nebulized budesonide (Pulmicort Respules/Nebuamp), oral prednisolone, and placebo was compared in 199 patients with acute exacerbations of COPD requiring hospitalization. Patients received from randomization (H_0) to 72 h (H_{72}), 2 mg of budesonide every 6 h (n = 71), 30 mg of oral prednisolone every 12 h (n = 62), or placebo (n = 66). All received standard treatment, including nebulized $ß_2$-agonists, ipratropium bromide, oral antibiotics, and supplemental oxygen. The mean change (95% confidence interval) in postbronchodilator FEV_1 from H_0 to H_{72} was greater with active treatments than with placebo: budesonide versus placebo, 0.10 L (0.02 to 0.18 L); prednisolone versus placebo, 0.16 L (0.08 to 0.24 L). The difference in FEV_1 between budesonide and prednisolone was not significant, -0.06 L (-0.14 to 0.02 L). The occurrence of serious adverse events was similar for all groups. Budesonide had less systemic activity than prednisolone as indicated by a higher incidence of hyperglycemia observed with prednisolone. Both budesonide and prednisolone improved airflow in COPD patients with acute exacerbations when compared with placebo. Nebulized budesonide may be an alternative to oral prednisolone in the treatment of nonacidotic exacerbations of COPD but further studies should be done to evaluate its long-term impact on clinical outcomes after an initial episode of COPD exacerbation.

The Impact of Chronic Obstructive Pulmonary Disease on Work Loss in the United States—Sin DD, Stafinski T, Ng YC, Bell NR, Jacobs P. Am J Respir Crit Care Med 2002 Mar 1;165(5):704-707.

Chronic obstructive pulmonary disease (COPD) is a rapidly growing public health problem in the United States and elsewhere. Although direct costs of COPD are well documented, the impact of COPD and its severity on labor force participation is not well known. Using population-based data from the Third National Health and Nutrition Examination Survey (NHANES III), we determined the adjusted relationship between COPD (and its severity) and labor force participation in the U.S. We used data from 12,436 participants involved in NHANES III; 1,073 of these participants (8.6% of the total) reported COPD. These participants were 3.9% (95% confidence interval, 1.3% to 6.4%) less likely to be in the labor force than those without COPD. Increasing severity of COPD was associated with decreased probability of being in the labor force (p for linear trend = 0.001). Mild, moderate, and severe COPD was associated with a 3.4%, 3.9%, and 14.4% reduction in the labor force participation rate relative to those without COPD. These data suggest that COPD has a considerable adverse impact on work force participation. Based on these data, we estimate that, in 1994, COPD was responsible for work loss of approximately $9.9 billion in the U.S.

Lung Cancer, Cardiopulmonary Mortality, and Long-Term Exposure to Fine Particulate Air Pollution—Pope CA 3rd, Burnett RT, Thun MJ, Calle EE, Krewski D, Ito K, Thurston GD. JAMA 2002 Mar 6;287(9):1132-1141.

CONTEXT: Associations have been found between day-to-day particulate air pollution and increased risk of various adverse health outcomes, including cardiopulmonary mortality. However, studies of health effects of long-term particulate air pollution have been less conclusive. OBJECTIVE: To assess the relationship between long-term exposure to fine particulate air pollution and all-cause, lung cancer, and cardiopulmonary mortality. DESIGN, SETTING, AND PARTICIPANTS: Vital status and cause of death data were collected by the American Cancer Society as part of the Cancer Prevention II study, an ongoing prospective mortality study, which enrolled approximately 1.2 million adults in 1982. Participants completed a questionnaire detailing individual risk factor data (age, sex, race, weight, height, smoking history, education, marital status, diet, alcohol consumption, and occupational exposures). The risk factor data for approximately 500 000 adults were linked with air pollution data for metropolitan areas throughout the United States and combined with vital status and cause of death data through December 31, 1998. MAIN OUTCOME MEASURE: All-cause, lung cancer, and cardiopulmonary mortality. RESULTS: Fine particulate and sulfur oxide—related pollution were associated with all-cause, lung cancer, and cardiopulmonary mortality. Each 10-microg/m^3 elevation in fine particulate air pollution was associated with approximately a 4%, 6%, and 8% increased risk of all-cause, cardiopulmonary, and lung cancer mortality, respectively. Measures of coarse particle fraction and total suspended particles were not consistently associated with mortality. CONCLUSION: Long-term exposure to combustion-related fine particulate air pollution is an important environmental risk factor for cardiopulmonary and lung cancer mortality.

Corticosteroid Therapy in Pulmonary Sarcoidosis: A Systematic Review—Paramothayan S, Jones PW. JAMA 2002 Mar 13;287(10):1301-1307.

CONTEXT: Corticosteroids are used in pulmonary sarcoidosis to reduce symptoms and minimize long-term damage. Spontaneous recovery is a common feature. Both the decision to initiate therapy and the treatment response may be influenced by disease severity, so trials need to use a randomized controlled design. OBJECTIVE: To assess the effect of oral and inhaled corticosteroids on chest radiograph results, symptoms, pulmonary function, and long-term outcome in pulmonary sarcoidosis. DATA SOURCES: MEDLINE, EMBASE, CINAHL, and the Cochrane Controlled Trials Register were searched all years through December 2001. Bibliographies of review articles and retrieved articles were searched, and pharmaceutical companies and authors of identified trials were contacted for other studies. There was no language restriction. STUDY SELECTION: Trials were randomized and included a control group. Participants were adults with histologic evidence of pulmonary sarcoidosis. Treatments included the use of oral and inhaled corticosteroids for at least 8 weeks. The search identified 150 studies; 9 met the inclusion criteria, but only 8 provided usable data. DATA EXTRACTION: Two reviewers assessed trial quality using the Jadad score, which evaluates the quality of randomization, blinding, and reasons for withdrawal. Data were extracted and sent to primary authors for verification. DATA SYNTHESIS: In patients with stage 2 and 3 disease, oral corticosteroids improved findings on the chest radiograph after 6 to 24 months (Peto odds ratio, 2.54; 95% confidence interval [CI], 1.69-3.81; p<0.001). Forced vital capacity improved with oral corticosteroids (weighted mean difference [WMD], 4.2% predicted; 95% CI, 0.4%-7.9% predicted) and diffusing capacity also improved (WMD, 5.7% predicted; 95% CI, 1.0%-10.5% predicted). In 2 small studies of inhaled corticosteroids, there was no effect on chest radiograph and inconsistent effects on lung function in one and only a small improvement in symptoms in the other. There were no data following corticosteroid withdrawal to assess any disease-modifying effect. CONCLUSIONS: Oral corticosteroids improved results on the chest radiograph following 6 to 24 months of treatment and produced a small improvement in vital capacity and diffusing capacity. Trials of inhaled corticosteroids were small and results too inconsistent to make firm conclusions concerning their efficacy. There are no data to suggest that corticosteroid therapy alters long-term disease progression.

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Serological Evidence of Legionella Species Infection in Acute Exacerbation of COPD—Lieberman D, Lieberman D, Shmarkov O, Gelfer Y, Ben-Yaakov M, Lazarovich Z, Boldur I. Eur Respir J 2002 Mar;19(3):392-397.

A prospective study was conducted to identify and characterize hospitalizations for acute exacerbations of chronic obstructive pulmonary disease (AECOPD) with serological evidence of infection with Legionella spp. (Lsp). Two-hundred and forty hospital admissions for AECOPD of 213 patients were included in the study. Paired sera were obtained for each of the admissions and were tested for 41 different serogroups of Lsp, using microimmunofluorescence-serology. Only a significant change in immunoglobulin-G and/or immunoglobulin-M antibody titres was considered diagnostic. In 40 admissions (16.7%) there was serological evidence of infection with Lsp (LspH). *Legionella pneumophila* 1 was identified in nine admissions, *L. pneumophila* 3-15 in 19 and nonpneumophila in 22. In 26 LspH (65%) there was serological evidence of infection with at least one other respiratory pathogen. Compared to the 200 admissions without Lsp (NLspH), the LspH patients were younger ($p<0.05$) and more hypoxaemic ($p<0.04$). None of the cases in the LspH group had an abrupt onset of disease, compared to 58 (29.0%) in the NLspH group ($p<0.0001$). The incidence of myalgia/arthralgia was 55% for LspH compared to 37% for NLspH ($p<0.03$). To conclude, serological evidence of infection with Legionella spp. is common among patients hospitalized with acute exacerbations of chronic obstructive pulmonary disease. In most hospital admissions with serological evidence of infection with Legionella spp. an additional respiratory pathogen can be identified. Acute exacerbation develops gradually in these patients and is characterized clinically by more systemic manifestations than hospital admissions without serological evidence of infection with Legionella spp. The true interpretation and practical relevance of these findings should be determined in further studies.

Interpreting Thresholds for a Clinically Significant Change in Health Status in Asthma and COPD—Jones PW. Eur Respir J 2002 Mar;19(3):398-404.

Health status (or Health-Related Quality of Life) measurement is an established method for assessing the overall efficacy of treatments for asthma and chronic obstructive pulmonary disease (COPD). Such measurements can indicate the potential clinical significance of a treatment's effect. This paper is concerned with methods of estimating the threshold of clinical significance for three widely used health status questionnaires for asthma and COPD: the Asthma Quality of Life Questionnaire, Chronic Respiratory Questionnaire and St George's Respiratory Questionnaire. It discusses the methodology used to obtain such estimates and shows that the estimates appear to be fairly reliable; ie. for a given questionnaire, similar estimates may be obtained in different studies. These empirically derived thresholds are all mean estimates with confidence intervals around them. The presence of these confidence intervals affects the way in which the thresholds may be used to draw inferences concerning the clinical relevance of clinical trial results. A new system of judging the magnitude of clinically significant results is proposed. Finally, an attempt is made to translate these thresholds into scenarios that illustrate what a clinically significant change with treatment may mean to an individual patient.

Interpretation of Quality of Life Scores from the St George's Respiratory Questionnaire—Ferrer M, Villasante C, Alonso J, Sobradillo V, Gabriel R, Vilagut G, et al. Eur Respir J 2002 Mar;19(3):405-413.

The aim of the study was to obtain the general population norms for the St. George's Respiratory Questionnaire (SGRQ), a specific questionnaire for respiratory diseases. The IBERPOC project was a cross-sectional study of representative samples of the general population aged between 40-69 yrs. The study sample was composed of 862 individuals. All participants considered as "probable cases" of chronic obstructive pulmonary disease (COPD) (n=460) were eligible to complete the SGRQ and among the rest of the nonprobable COPD participants (n=3,571), 10 individuals from each defined age and sex group were eligible (n=402). Weights were applied to restore general population representativity of the sample. Mean forced expiratory volume in one second (FEV_1) predicted was 89.4% (SD=16.5%; range: 16-131%). Chronbach's α coefficients were > 0.7 in the symptoms, activity and impact scales, and >0.9 in the overall scale. Symptom scale score was significantly higher among males (11.6 versus 7.8; $p<0.01$) and activity scale score was significantly higher among females (12.2 versus 14.6; $p=0.04$). In a multiple linear regression model, respiratory diseases (asthma and COPD) and FEV_1 % over pred showed the strongest association with the SGRQ total score. Smoking, sex, age and education were independently associated with the total SGRQ score. These results indicate that individuals from the general population presented some of the problems that are important when measuring health-related quality of life in respiratory patients, and provide St George's Respiratory Questionnaire norms, a useful method for interpreting the St George's Respiratory Questionnaire score in a given patient or study samples.

Effect of Chest Wall Vibration on the Canine Diaphragm During Breathing—Leduc D, De TA. Eur Respir J 2002 Mar;19(3):429-433.

High-frequency mechanical vibration of the ribcage reduces dyspnoea in patients with chronic obstructive pulmonary disease, and the suggestion has been made that this effect might be related to a decrease in central respiratory drive resulting from an increase in afferent inputs from intercostal muscles. In the present studies, the effects of ribcage vibration on central respiratory drive have been assessed without the confounding influence of conscious reactions. The electromyographic (EMG) activity of the diaphragm and the changes in pleural (Ppl) and abdominal (Pab) pressure were measured in six anaesthetized, spontaneously-breathing dogs while the rostral, the middle, or the caudal portion of the ribcage was vibrated at intervals during inspiration. The EMG activity of the external and parasternal intercostals was also measured. Ribcage vibration consistently elicited a marked increase in the inspiratory EMG activity recorded from the external intercostals, thus indicating that the procedure did activate intercostal muscle spindles. However, no alteration in diaphragmatic or parasternal intercostal EMG activity was seen in any animal. Transdiaphragmatic pressure and the relationship between ΔPab and ΔPpl during inspiration were also unaltered. The authors conclude that ribcage vibration and, with it, stimulation of external intercostal muscle spindles has no significant influence on phrenic motoneurones or on medullary inspiratory neurones. It is unlikely, therefore, that the beneficial effect of the procedure on dyspnoea results from a specific reduction in central respiratory drive.

Relationship Between Supramaximal Flow During Cough and Mortality in Motor Neurone Disease—Chaudri MB, Liu C, Hubbard R, Jefferson D, Kinnear WJ. Eur Respir J 2002 Mar;19(3):434-438.

The main function of cough is clearance of intrathoracic airways. A normal cough is characterized by a transient increase in expiratory flow above the maximal flow-volume loop envelope, known as cough "spikes". They may be absent in patients with motor neurone disease. The relationship between cough pattern, pulmonary function and survival was studied. Fifty-three patients were recruited (25 bulbar). Vital capacity, maximal inspiratory and expiratory mouth pressures and cough flow/volume curves were performed on all patients, and the presence or absence of spikes were recorded. The primary endpoints were mortality or initiation of ventilatory support over a period of 18 months. Thirty-five patients died over the 18-month period of the study (including the six who were started on noninvasive ventilation). Twelve of the 24 patients

with spikes died compared to 23 out of 29 patients without spikes ($p<0.05$). Patients without spikes were more likely to be bulbar on clinical grounds ($p<0.0001$) and had poorer lung function. The results showed an association between the absence of cough spikes and increased mortality. However the main determinants of survival in motor neurone disease are age, vital capacity and inspiratory mouth pressure, and it remains to be shown whether regular monitoring of cough conveys any additional advantage.

Tidal Expiratory Flow Limitation and Chronic Dyspnoea in Patients with Cystic Fibrosis—Goetghebeur D, Sarni D, Grossi Y, Leroyer C, Ghezzo H, Milic-Emiri J, Bellet M. Eur Respir J 2002 Mar;19(3):492-498.

Cystic fibrosis (CF) eventually leads to hyperinflation linked to tidal expiratory flow limitation (FL) and ventilatory failure. Presence of FL was assessed at rest in 22 seated children and adults with CF (forced expiratory volume in one second (FEV_1) range: 16-92% predicted), using both the negative expiratory pressure (NEP) technique and the "conventional" method based on comparison of tidal and maximal expiratory flow/volume curves. In addition, chronic dyspnoea was scored with the modified Medical Research Council (MRC) scale. Measurements were made before and 15 min after inhalation of salbutamol. With NEP, FL was present in only three malnourished patients, who had the lowest FEV_1 values (16-27% pred) and claimed very severe dyspnoea (MRC score 5). By contrast, an additional seven patients were classified as FL with the conventional method. Six of these patients had little or no dyspnoea (MRC scores 0-1). Salbutamol administration had no effect on the extent of FL, and the concomitant decrease in functional residual capacity (FRC) was too small to play any clinically significant role. This study concluded that in seated patients with cystic fibrosis, expiratory flow limitation is absent at rest, unless the forced expiratory volume in one second is <30% predicted. If present, expiratory flow limitation is associated with severe chronic dyspnoea. The conventional method for assessing expiratory flow limitation is not reliable and bronchodilator administration has little effect on expiratory flow limitation.

Health-Related Quality of Life in Obstructive Sleep Apnoea—Lacasse Y, Godbout C, Series F. Eur Respir J 2002 Mar;19(3):499-503.

The identification of the areas of quality of life (QoL) most likely to be affected by obstructive sleep apnoea (OSA) would be an important step in the evaluation of the impact of the disease and its treatment modalities. The objective of this study was to describe the impact of OSA on patients' QoL. A list of 186 items potentially related to QoL of patients with OSA was constructed. From this list, consecutive patients were asked, at the time of the diagnosis, to identify the most significant items and to grade their importance on a 5-point scale. The item impact was determined from the proportion of patients who identified it as important, and the mean importance score attributed to this item (impact score=frequency x importance). One hundred patients (82 male; mean age: 51 yrs) were interviewed. The items having the most important impact on QoL clustered into five domains: 1) daytime symptoms; 2) nocturnal symptoms; 3) limitation of activities; 4) emotions; and 5) interpersonal relationships. The impact of obstructive sleep apnoea on quality of life is not limited to excessive daytime sleepiness. Obstructive sleep apnoea significantly contributes to the impairment of all domains of what is usually referred to as "health-related quality of life."

Sleep Quality and Daytime Function in Adults with Cystic Fibrosis and Severe Lung Disease—Dancey DR, Tullis ED, Heslegrave R, Thornley K, Hanly PJ. Eur Respir J 2002 Mar;19(3):504-510.

It was hypothesized that adult cystic fibrosis (CF) patients with severe lung disease have impaired daytime function related to nocturnal hypoxaemia and sleep disruption. Nineteen CF patients (forced expiratory volume in one second 28±7% predicted) and 10 healthy subjects completed sleep diaries, overnight polysomnography (PSG), and assessment of daytime sleepiness and neurocognitive function. CF patients tended to report more awakenings (0.7±0.5 versus 0.3±0.2 x h^{-1}, p=0.08), and PSG revealed reduced sleep efficiency (71±25 versus 93±4%, p=0.004) and a higher frequency of awakenings (4.2±2.7 versus 2.4±1.4 x h^{-1}, p=0.06). Mean arterial oxygen saturation during sleep was lower in CF patients (84.4±6.8 versus 94.3±1.5%, $p<0.0001$) and was associated with reduced sleep efficiency (regression coefficient (r)=0.57, p=0.014). CF patients had short sleep latency on the multiple sleep latency test (6.7±3 min). The CF group reported lower levels of activation and happiness and greater levels of fatigue ($p<0.01$), which correlated with indices of sleep loss, such as sleep efficiency (r=0.47, p=10.05). Objective neurocognitive performance was also impaired in CF patients, reflected by lower throughput for simple addition/subtraction, serial reaction and colour-word conflict. The authors concluded that adult cystic fibrosis patients with severe lung disease have impaired neurocognitive function and daytime sleepiness, which is partly related to chronic sleep loss and nocturnal hypoxaemia.

Aerosolized Iloprost Induces a Mild but Sustained Inhibition of Platelet Aggregation—Beghetti M, Reber G, de MP, Vadas L, Chiappe A, Spahr-Schopfer I, Rimensberger PC. Eur Respir J 2002 Mar;19(3):518-524.

Pathological studies have revealed that one of the main features encountered in the pulmonary vasculature of patients with pulmonary hypertension is the presence of thrombotic lesions. Open pilot studies have indicated that aerosolized iloprost may have beneficial effects in patients with pulmonary hypertension. The effects of aerosolized iloprost on platelet function and plasma cyclic adenosine monophosphate (cAMP) were studied. Platelet aggregation and plasma cAMP were measured at baseline, 30 min, 4 and 6 h after inhalation of 15 microg iloprost in 10 healthy volunteers. Maximal platelet aggregation in response to adenosine diphosphate (ADP) (2 and 6 micromol x L^{-1}), collagen (2.5 and 5 microg x mL^{-1}), epinephrine (1.25 and 5 micromol x L^{-1}) and arachidonic acid (0.5 mg x mL^{-1}) was measured. Platelet aggregation was significantly inhibited at 30 min in response to ADP (2 and 6 micromol x L^{-1}, epinephrine (1.25 and 5 micromol x L^{-1}) and collagen (2.5 microg x mL^{-1}). It was still inhibited at 4 h in response to the same agents, but normalized at 6 h. cAMP increased at 30 min, from 27.3±1.2 to 31.8±1.2 nmol x L^{-1}, remained increased at 4 h (29.2±1.3 nmol x L^{-1}) and normalized at 6 h (27.4±1.1 nmol x L^{-1}). Aerosolized iloprost induced a mild but sustained inhibition of platelet aggregation. Platelet aggregation inhibition may be one of the mechanisms which explains the beneficial effect of repeated inhalation of iloprost in pulmonary hypertension.

Recording Flow in the First Second of a Maximal Forced Expiratory Manoeuvre: Influence of Frequency Content—Miller MR, Lloyd J, Bright P. Eur Respir J 2002 Mar;19(3):530-533.

The frequency content of the first second of the maximum forced expiratory manoeuvre (MFEM) was measured to determine if the currently accepted frequency limit of 20 Hz for MFEM is adequate for recording peak expiratory flow (PEF). The frequency response of a Fleisch pneumotachograph (PT) was measured and used to record MFEM from 24 patients attending a lung-function laboratory and 26 normal volunteers. The first 1.024 s of the signal recorded at 1,000 Hz for that blow with maximum PEF, underwent fast Fourier transformation using a triangular window function, applied after 0.75 s to reduce flow linearly to zero. All the frequencies above a set limit were removed, followed by inverse transformation to reconstitute the blow. The limits for this frequency cut-off were progressively varied from 100 Hz down to 15 Hz, with the resulting PEF being compared with the PEF from the reconstituted blow with no frequency reduction. The average±SD age for the group was

47±18 yrs and the average PEF was 450±187 L x min^{-1}, which, when expressed as a standardized residual, was 0.1±2.1, with a range from -4.5-3.9 indicating a good spread around normal values. Average rise time to PEF was 83±38 ms and dwell time >90% PEF was 45±25 ms. Cut-off >20 Hz reduced the mean PEF of the group by 8.5 L x min^{-1} (95% confidence limit 5.5-11.4 L x min^{-1}), whereas cut-off >30 Hz reduced mean PEF by 4.4 L x min^{-1} (2.6-6.2). In the present study subjects, 30 Hz was on the 95th percentile of frequencies for defining the upper limit for 98% of the power spectrum for the first second of the blow. It has been shown that there are frequencies >20 Hz that contribute to peak expiratory flow enough to influence readings made using conventional hand-held peak expiratory flow meters, such as the mini-Wright. Devices used for recording flow from a maximum forced expiratory manoeuvre should therefore have an adequate frequency response of up to 30 Hz.

Changes in the Transmission of Tuberculosis in New York City from 1990 to 1999—Geng E, Kreiswirth B, Driver C, Li J, Burzynski J, DellaLatta P, et al. N Engl J Med 2002 May 9;346(19):1453-1458.

BACKGROUND: Over the past decade, there has been a reduction in the incidence of tuberculosis in New York City and in the United States. However, the reduction has been confined mainly to U.S.-born persons. Understanding the reasons for the lack of reduction among non-U.S.-born persons may lead to new strategies for tuberculosis control. METHODS: We performed DNA fingerprinting with the IS6110 insertion sequence of the organisms isolated from patients with culture-positive tuberculosis in northern Manhattan from 1990 to 1999. The goal was to identify the strains responsible for multiple infections, presumably through recent transmission (clusters of cases), as well as the strains found in only one patient, presumably representing reactivation of latent infection. RESULTS: Of 546 available isolates of *Mycobacterium tuberculosis*, 261 (48 %) belonged to a cluster and 285 (52 %) did not. In multivariate analysis, significant predictors of noncluster status included birth outside the United States (odds ratio for a strain causing a cluster among non-Hispanic foreign-born patients, 0.31; 95 % confidence interval, 0.14 to 0.66; odds ratio among Hispanic foreign-born patients, 0.51; 95 % confidence interval, 0.30 to 0.88), age > 60 years (odds ratio, 0.37), and diagnosis after 1993 (odds ratio, 0.50). All these characteristics appeared to be associated with reactivation disease rather than with tuberculosis due to recent transmission. Homelessness was associated with clustering (odds ratio, 1.78; 95 % confidence interval, 0.99 to 3.20) and therefore with recent transmission. CONCLUSIONS: These findings from northern Manhattan suggest that among foreign-born persons, tuberculosis is largely caused by reactivation of latent infection, whereas among U.S.-born persons, many cases result from recent transmission. Strategies for the control and elimination of tuberculosis among foreign-born persons at high risk should be directed toward the treatment of latent tuberculosis infection.

Benefit of Atrial Pacing in Sleep Apnea Syndrome—Garrigue S, Bordier P, Jais P, Shah DC, Hocini M, Raherison C, et al. N Engl J Med. 2002 Feb 7;346(6):444-445. *Erratum in: N Engl J Med 2002 Mar 14;346(11):872.*

BACKGROUND: Many patients with sleep apnea syndrome have nocturnal bradycardia, paroxysmal tachyarrhythmias, or both, which can be prevented by permanent atrial pacing. We evaluated the effect of using cardiac pacing to increase the heart rate during sleep in patients with sleep apnea syndrome. METHODS: We studied 15 patients (11 men and 4 women; mean [±SD] age, 69±9 years) with central or obstructive sleep apnea who had received permanent atrial-synchronous ventricular pacemakers for symptomatic sinus bradycardia. All patients underwent three polysomnographic evaluations on consecutive nights, the first night for base-line evaluation and then, in random order, one night in spontaneous rhythm and one in dual-chamber pacing mode with atrial overdrive (basic rate, 15 beats per minute faster than the mean nocturnal sinus rate). The total duration and number of episodes of central or obstructive sleep apnea or hypopnea were analyzed and compared. RESULTS: The mean 24-hour sinus rate during spontaneous rhythm was 57 ± 5 beats per minute at base line, as compared with 72 ± 3 beats per minute with atrial overdrive pacing ($p<0.001$). The total duration of sleep was 321 ± 49 minutes in spontaneous rhythm, as compared with 331 ± 46 minutes with atrial overdrive pacing ($p=0.48$). The hypopnea index (the total number of episodes of hypopnea divided by the number of hours of sleep) was reduced from 9 ± 4 in spontaneous rhythm to 3 ±3 with atrial overdrive pacing ($p<0.001$). For both apnea and hypopnea, the value for the index was 28 ± 22 in spontaneous rhythm, as compared with 11 ± 14 with atrial overdrive pacing ($p<0.001$). CONCLUSIONS: In patients with sleep apnea syndrome, atrial overdrive pacing significantly reduces the number of episodes of central or obstructive sleep apnea without reducing the total sleep time

Strategies for Culturally Effective End-of-Life Care—Crawley LM, Marshall PA, Lo B, Koenig BA; The End-of-Life Care Consensus Panel. Ann Intern Med 2002 May 7;136(9):673-679.

As a result of profound worldwide demographic change, physicians will increasingly care for patients from cultural backgrounds other than their own. Differences in beliefs, values, and traditional health care practices are of particular relevance at the end of life. Health care providers and patients and families may not have shared understandings of the meaning of illness or death and may not agree on the best strategies to plan for the end of life or to alleviate pain and suffering. Good end-of-life care may be complicated by disagreements between physicians and patients, difficult interactions, or decisions the physician does not understand. Challenges may result from cultural differences between the patient's background and traditional medical practice. Values so ingrained in physicians as to be unquestioned may be alien to patients from different backgrounds. Physicians need to be sensitive to cultural differences and to develop the skills necessary to work with patients from diverse backgrounds. Community and cultural ties provide a source of great comfort as patients and families prepare for death. This paper describes two cases that raise issues about cross-cultural end-of-life practice and suggests strategies for negotiating common problems. Physicians should assess the cultural background of each patient and inquire about values that may affect care at the end of life. They should become aware of the specific beliefs and practices of the populations they serve, always remembering to inquire whether an individual patient adheres to these cultural beliefs. Attention to cultural difference enables the physician to provide comprehensive and compassionate palliative care at the end of life.

Transition Programs in Cystic Fibrosis Centers: Perceptions of Patients—Anderson DL, Flume PA, Hardy KK, Gray S. Pediatr Pulmonol 2002 May;33(5):327-331.

There is a growing population of adults with cystic fibrosis (CF) and a need for development of adult CF programs. Recommendations for transfer of patients from pediatric care to an adult program include a transition program. The purpose of this study was to survey adult CF patients to assess their own concerns regarding this issue. A survey was sent to all 1,288 members of the International Association of Cystic Fibrosis Adults (IACFA), with a response rate of 25.9% (n = 334). The majority of patients (81.2%) received care from a CF center; the major difference between those seen at a CF center and those seen at another facility was proximity to a CF center. Nearly one-fourth of patients seen at a CF center continued to receive care from a pediatrician even though a CF-trained internist was available; though these patients were younger, their mean age was still about 30 years. Patients seen by a pediatrician were more like to be students and to live with their parents. Those patients seen in an

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adult program described a variety of criteria for their transfer to the adult pro-gram, but there were no consistent findings to suggest a standard transition program. Indeed, many patients did not meet the adult team until the time of the transfer. Most importantly, the patients reported their level of concern about transfer as minimal, far less than what CF physicians had perceived. These differences may impede the successful transition of patients into an adult program.

Correlation Between Digital Clubbing and Pulmonary Function in Cystic Fibrosis—Nakamura CT, Ng GY, Paton JY, Keens TG, Witmer JC, Bautista-Bolduc D, Woo MS. Pediatr Pulmonol 2002 May;33(5):332-338.

The correlation between digital clubbing and certain pulmonary function derangements (hypoxemia and FEV_1) was previously described. However, the relationship between digital clubbing and other measures of pulmonary function or the presence of liver disease in patients with cystic fibrosis (CF) is poorly defined. Hence we compared the digital clubbing index (CI: ratio of distal phalangeal depth to interphalangeal depth) of 100 patients with CF (43 males, 57 females; mean age, 15.7 ± 7.3 years) with that of 100 age- and gender-matched healthy controls. Digital clubbing was defined as a CI ≥ 1.00 (mean ± 2.6 SD; 99% of normal subjects). The CI and its relationship to pulmonary function and to liver disease was then evaluated in the CF patients. Digital clubbing was present in 75/100 (75%) of CF patients but was absent in all controls ($p < 0.0001$). In CF patients, CI was inversely correlated with P_{aO_2} ($r = -0.555$; $p < 0.001$), FEV_1 ($r = -0.499$; $p < 0.001$), and $FEF_{25-75\%}$ ($r = -0.404$; $p < 0.001$), and was positively correlated with RV ($r = 0.285$; $p < 0.05$) and the slope of phase 3 of single-breath nitrogen washout ($SP3N_2$) ($r = 0.532$; $p < 0.01$). There was no significant correlation between CI and age ($r = 0.020$; $p = 0.84$), TLC ($r = -0.097$; $p = 0.34$), P_{aCO_2} ($r = 0.167$; $p = 0.10$), or history of liver disease ($p = 0.08$). We conclude that in CF, the degree of digital clubbing is related to degree of hypoxemia, airways obstruction, hyperinflation, and nonuniformity of ventilation.

Subcutaneous Terbutaline in Children with Chronic Severe Asthma—Payne DN, Balfour-Lynn IM, Biggart EA, Bush A, Rosenthal M. Pediatr Pulmonol 2002 May;33(5):356-361.

A continuous subcutaneous infusion of terbutaline (CSIT) was used to treat 8 children with chronic severe asthma who continued to experience frequent symptoms, despite treatment with regular oral prednisolone. Five patients experienced a symptomatic improvement from CSIT, leading to a reduction in regular medication. Three patients did not experience any lasting benefit from CSIT. The most common side effects were related to the infusion site (bruising and local infection). CSIT may lead to an improvement in symptoms and a reduction in oral steroid dose in selected children with chronic severe asthma. These initial findings support the need for further controlled studies to evaluate the use of CSIT in severe childhood asthma.

Effects of Intrabronchial Foreign Body Retention—Yildizeli B, Zonuzi F, Yuksel M, Kodalli N, Cakalagaoglu F, Kullu S. Pediatr Pulmonol 2002 May;33(5):362-367.

Unrecognized bronchial foreign bodies (Fbs) cause irreversible changes in the airways. However, the exact course of these changes is not well-known. We developed an animal model of bronchial obstruction to radiologically and histopathologically assess the development of postobstructive pulmonary changes. A piece of peanut was placed in the airways of 21 rabbits through a 2.5-mm rigid bronchoscope. Animals were divided into three groups (groups I-III) that were sacrificed on day 3,10, and 30 after Fb placement, respectively. Prior to sacrifice, since there were no differences between the groups prior to Fb placement, computerized tomography (CT) of the lung was taken, and the lungs were harvested for histologic analysis under light microscope. In group I, leukocyte infiltration around the bronchial wall ($p = 0.0003$) and edema ($p = 0.0384$) around the alveolar septa were the predominant histological findings. The CT scan was normal. In group II and group III, increased amounts of mononuclear cells and macrophage infiltration around the bronchial wall were observed ($p = 0.0008$, $p = 0.0409$, respectively). There were no differences in presence of granuloma formation, emphysema, atelectasis, or thickness of alveolar septa among the three groups. The CT scan of group II showed consolidations plus minimal bronchial dilatation in the involved lung of the rabbits (p not significant). Bronchial cartilage destruction was seen in 4 out of 7 rabbits in group III ($p = 0.0071$). We conclude that 30-day retention of intrabronchial peanut caused bronchial cartilage destruction and fibrosis that were attributed as bronchiectatic changes in the airways of the lung parenchyma. Therefore, any case with suspected foreign body aspiration should be treated immediately to prevent possible irreversible changes of the lungs.

Noninvasive Investigation of Hepatopulmonary Syndrome in Children and Adolescents with Chronic Cholestasis—Santamaria F, Sarnelli P, Celentano L, Farina V, Vegnente A, Mansi A, et al. Pediatr Pulmonol 2002 May;33(5):374-379.

Early detection of hepatopulmonary syndrome (HPS) may be delayed because of invasiveness of the diagnostic procedures. In this pilot study, we prospectively investigated the usefulness of determining transcutaneous O_2 tension after 100% O_2 ($TcPO_2100$) breathing using a transcutaneous hyperoxia test (THT) in 11 children with chronic cholestasis and without primary cardiopulmonary disease. These patients also underwent alveolar-arterial O_2 gradient testing ($AaDO_2$) at an inspired oxygen fraction (F_{IO_2}) of 0.21, lung scintiscan, and contrast transthoracic echocardiography (TTE). Three of them had a liver transplantation because of the downhill course of their liver disease and respiratory status. THT transcutaneous O_2 tension at 21% F_{IO_2} ($TcPO_221$) was 75 ± 13 mmHg, and increased to 488 ± 106 mm Hg after 100% O_2 breathing ($TcPO_2100$). Both mean values were not significantly different from those found in 8 age-matched controls ($p = 0.9$ and $p = 0.5$, respectively). However, one patient, in spite of her stable liver function, showed an abnormal $TcPO_221$ and $TcPO_2100$ (45 mm Hg and 210 mm Hg, respectively). This same subject was also the only patient with abnormalities of $AaDO_2$ (54.2 mm Hg; normal value, < 20 mm Hg), lung scintiscan (brain/lung ratio of technetium-99 fixation (B/L SI) = 9, normal value < 1), and TTE, suggesting intrapulmonary vasodilatations and shunts. Given the clinical development of cyanosis and platypnea, all criteria for HPS were fulfilled, and timing of her liver transplantation was therefore accelerated. This resulted in HPS regression. In children with chronic cholestasis, repeated transcutaneous bedside measurements are a rapid and reliable noninvasive test for characterizing the severity of abnormal oxygenation, and may prove useful also in liver posttransplantation monitoring.

Assessment of Inspiratory Flow Limitation in Children with Sleep-Disordered Breathing by a Nasal Cannula Pressure Transducer System—Serebrisky D, Cordero R, Mandeli J, Kattan M, Lamm C. Pediatr Pulmonol 2002 May;33(5):380-387.

A nasal cannula pressure transducer system identifies inspiratory flow limitation and increased upper airway resistance in adults with sleep-disordered breathing (SDB). The purpose of this study was to evaluate whether nasal cannula pressure (NCP) detects apneas and hypopneas as well as additional flow-limited events associated with increased airway resistance in children. We studied NCP in 47 patients (ages 2-14 years) referred for SDB to a university-based sleep disorders program during nocturnal polysomnography (NPSG). During NPSG, airflow was assessed simultaneously by thermistor and NCP. There was a high correlation between apneas assessed by thermistor (T) and NCP ($r = 0.90$, $p < 0.0001$), and for hypopneas using these two methods ($r = 0.94$, $p =$

0.0001). Respiratory driving pressure was indirectly measured with an esophageal pressure catheter. Flow-limited (flattened) NCP waves were associated with significantly higher driving pressure, indicating elevated upper airway resistance, compared to nonflow-limited (rounded) waves during nonrapid eye movement (NREM) ($p = 0.05$) and rapid eye movement (REM) ($p = 0.01$) sleep. Patients were classified as either having obstructive sleep apnea syndrome (OSAS) or primary snoring, based on standard NPSG criteria. NCP identified additional respiratory events with a flattened contour (FC) not detected by thermistor. NCP is a noninvasive device that identifies obstructive apneas and hypopneas as well as additional respiratory events associated with flow limitation in children.

Assessment of a Low-Cost Home Monitoring Spirometer for Children—Bastian-Lee Y, Chavasse R, Richter H, Seddon P. Pediatr Pulmonol 2002 May;33(5):388-394.

Electronic devices are now available to measure and store lung function parameters in the home. Before adopting a device for clinical or research use, it is important to validate it in the target patient group. The aim of this study was to assess a low-cost, portable, logging spirometer, the VM Plus (VM), against a standard laboratory Jaeger spirometer (JS) for use in children with respiratory disease. Seventy children with stable asthma or cystic fibrosis performed spirometry on the two devices, and results for peak expiratory flow (PEF) and forced expiratory volume in 1 sec (FEV_1) were compared. Comparison was made both using the two devices separately (separate method) and with the devices connected in series (series method). Reproducibility of the VM measurements was also assessed. Correlation between measurements was close (R values: separate, PEF, 0.91; FEV_1, 0.94; series, PEF, 0.97, FEV_1, 0.99), but PEF readings on the VM Plus were substantially higher than with the JS (mean difference: separate, 54.8 L/min; series, 28.2 L/min). This reflects well-reported differences in PEF measurements between the Mini-Wright PEF meter, on which the VM Plus spirometer is based, and conventional spirometers. Limits of agreement (series method) were: PEF, -13.2 to +69.6 L/min; FEV_1, -0.03 to +0.19 L. Reproducibility of VM Plus measurements was acceptable: coefficient of variation for PEF was 4%; for FEV_1, 4.3%; coefficient of reproducibility for PEF, 39 L/min; for FEV_1, 0.26 L. The VM Plus provides reasonably accurate, reproducible measurements of PEF and FEV_1, but intrinsic bias, particularly in PEF measurement, needs to be taken into account. Its potential to document longitudinal changes in lung function in children with respiratory disease at home merits further study.

Two-Year Retrospective Economic Evaluation of Three Dual-Controller Therapies Used in the Treatment of Asthma—O'Connor RD, O'Donnell JC, Pinto LA, Wiener DJ, Legorreta AP. Chest 2002 Apr;121(4):1028-1035.

OBJECTIVE: To compare asthma-related health-care utilization and expenditures for patients prescribed one of three dual-controller therapies: fluticasone plus salmeterol, inhaled corticosteroids (ICS) [excluding fluticasone] plus salmeterol, and ICS plus a leukotriene modifier (LTM). MATERIALS AND METHODS: Asthma-related medical claims from two major health plans were obtained for the 12 months before and after the initiation of dual therapy. A total of 1,325 patients ≥ 12 years old with no claims for COPD or respiratory tract cancer were selected from the approximately 3.5 million lives covered. Multivariable regression was used to assess differences in asthma-related expenditures. To compensate for positive skew, all cost variables were log-transformed. RESULTS: Risk-adjusted total asthma-related costs for the fluticasone-plus-salmeterol cohort (n = 121), the ICS-plus-salmeterol cohort (n = 844), and the ICS-plus-LTM cohort (n = 30) were $975, $1,089, and $1,268, respectively. Risk-adjusted pharmacy costs were $813, $841, and $996, respectively. Generalized linear modeling, controlling for baseline covariates, indicated that compared to ICS-plus-LTM therapy, fluticasone-plus-salmeterol therapy was associated with a significant reduction in asthma-related total ($p = 0.0014$) and pharmacy ($p = 0.001$) costs. Similar results were found when the ICS-plus-salmeterol group and the ICS-plus-LTM group were compared ($p = 0.0001$). The number of inpatient, outpatient, and emergency department visits and their corresponding costs were lower for the fluticasone-plus-salmeterol cohort, but were not statistically significant ($p > 0.05$). CONCLUSION: Results from managed-care practice suggest that treatment with fluticasone plus salmeterol, and more broadly ICS plus salmeterol, yield important cost savings when compared to treatment with ICS plus LTM.

A Comparison of Albuterol Administered by Metered-Dose Inhaler and Spacer with Albuterol by Nebulizer in Adults Presenting to an Urban Emergency Department with Acute Asthma—Newman KB, Milne S, Hamilton C, Hall K. Chest 2002 Apr;121(4):1036-1041.

STUDY OBJECTIVES: To determine the efficacy of albuterol by metered-dose inhaler (MDI) and spacer compared to a nebulizer. DESIGN: A prospective, open-label study. SETTING: Large urban emergency department (ED). PATIENTS: All consecutive adult asthma patients over a 2.5-year period. INTERVENTIONS: ED personnel used a standardized treatment algorithm, which included albuterol administered by nebulization, for patients presenting to the ED during the first 12 months of the study. The treatment algorithm then was switched to one that utilized albuterol administered by MDI/spacer as the primary mode of delivery for the following 18 months. As part of the conversion to MDI/spacer, ED staff counseled patients on self-management and supplied patients with a peak flowmeter, an MDI/spacer, and an inhaled steroid for home use. MEASUREMENTS: Pulmonary function, clinical outcome, laboratory data, and financial data were assembled and analyzed from 2,342 ED visits and 1,420 patients. RESULTS: While there was no significant difference in hospital admission rates between patients in the MDI/spacer group and the nebulizer group (13.2% and 14.6%, respectively), there was a statistically greater improvement in peak flow rates in the MDI/spacer group (126.8 vs 111.9 L/min, respectively; $p = 0.002$). The MDI/spacer group also spent significantly less time in the ED (163.6 and 175 min, respectively; $p = 0.007$), had a lower total albuterol dose (1,125 microg and 6,700 microg, respectively; $p < 0.001$), and showed a greater improvement in arterial oxygen saturation ($p = 0.043$). Relapse rates at 14 and 21 days were significantly lower ($p < 0.01$ and $p < 0.05$, respectively) among patients treated with the MDI/spacer and were associated with asthma education and the provision of a peak flowmeter, a spacer, and an inhaled corticosteroid for patients' home use. CONCLUSIONS: Albuterol administered by MDI/spacer is an efficacious and cost-effective alternative to nebulization in adults with acute asthma who present at a large urban ED.

Association of Malignancy with Diseases Causing Interstitial Pulmonary Changes—Bouros D, Hatzakis K, Labrakis H, Zeibecoglou K. Chest 2002 Apr;121(4):1278-1289.

A number of studies have shown a high incidence of lung cancer in patients with idiopathic pulmonary fibrosis (9.8 to 38%) compared to control subjects (2 to 6.4%). A similar trend occurs in other entities that affect the interstitial lung compartment, such as systemic sclerosis and sarcoidosis, as well as occupational diseases. The pathogenesis of lung cancer in patients with diffuse pulmonary fibrosis is still unclear. Recent progress in molecular and cellular biology has shed some light on the possible interactions of several types of inflammatory cells, following the deleterious effects of toxic factors leading to alveolitis, and destruction and disorganization of lung parenchyma, which results in fibrosis. Further research in the field would enhance our understanding of the pathogenic mechanisms of cancer development in these patients, and to explain the reason for the different incidence of lung cancer in patients with various interstitial lung diseases.

Pulmonary Gas Exchange During Sleep in Patients with Airflow Limitation Undergoing Long-Term Oxygen Therapy

Long-term oxygen therapy (LTOT) is a routine treatment for chronic respiratory failure. Worldwide, more than 2 million patients benefit from LTOT.[1] Although the indications for and methods of LTOT are well established,[2,3] there are aspects of LTOT that remain matters of ongoing debate.

One such subject of uncertainty is the effect of oxygen treatment during sleep. Sleep induces important changes in breathing.[4] Lack of cortical control, resetting of the respiratory center to higher P_{aCO_2}, and decreased intercostal muscle activity lead to alveolar hypoventilation. During sleep, retention of bronchial secretions, increased airway resistance, and decreased functional residual capacity aggravate ventilation-perfusion imbalance. Those changes unfavorably influence pulmonary gas exchange.

Robin was probably the first to suggest that hypoxemia is aggravated during sleep in patients with chronic obstructive pulmonary disease (COPD).[5] This was confirmed by Koo et al,[6] who performed serial measurements of arterial blood gases in sleeping COPD patients and found P_{aO_2} lower and P_{aCO_2} higher than in the awake state. The lowest P_{aO_2} and the highest P_{aCO_2} were observed during deep sleep and rapid-eye-movement sleep.

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The introduction of continuous, noninvasive measurement of blood oxygen saturation (via pulse oximetry [S_{pO_2}]) allowed for more accurate measurement during sleep.[7,8] Oximetry recordings demonstrated that patients with advanced COPD experience multiple, prolonged, (20–30-min) desaturation episodes, mostly during rapid-eye-movement sleep.

Those observations led to a hypothesis (forwarded independently on both sides of the Atlantic) that aggravation of hypoxemia (more precisely, aggravation of alveolar hypoxia) during sleep accelerates development of pulmonary hypertension (cor pulmonale) in COPD.[9,10]

Since then the importance of supplemental oxygen during sleep has been emphasized in all LTOT guidelines. It was assumed that oxygen supplementation during sleep would prevent nocturnal desaturations, but several researchers have found that nocturnal desaturations can occur in sleeping patients breathing oxygen at a dose that assures satisfactory oxygenation at rest during the day. Śliwiński et al[11] performed 24-hour pulse oximetry with at-home COPD/LTOT patients and found that the majority of patients had periods of substantial desaturation during sleep, spending > 2 hours of sleep with S_{pO_2} below 90%, despite breathing supplemental oxygen.

Plywaczewski et al[12] performed 2 overnight pulse oximetry measurements, one on air, the other on oxygen, with 82 nonselected, consecutive, COPD patients qualified for LTOT. Thirty-nine subjects (48%) spent 66% of the night with S_{pO_2} below 90%, despite breathing supplemental oxygen.

In this issue of Respiratory Care, Tárrega et al[13] point out another problem related to breathing oxygen during sleep. With 39 patients undergoing or qualified for LTOT, Tárrega et al measured arterial blood gases at 3:00 AM, 7:00 AM, and noon while the patients were breathing oxygen (mean flow of 1.4 L/min) sufficient to raise S_{pO_2} to ≥ 90%. In 23 "poor responders" there was significant P_{aCO_2} increase (> 10 mm Hg) and/or pH decrease (to < 7.33) during the night hours. In 16 "good responders" there was either no P_{aCO_2} increase or the increase was negligible.

Interpreting those findings is difficult. First, the P_{aCO_2} increase observed in the majority of studied subjects was unusually high. The P_{aCO_2} increase during sleep in normal humans does not exceed 3–4 mm Hg.[14] A similar P_{aCO_2} increase was observed in stable COPD patients.[6,15] The significant P_{aCO_2} increase during sleep was observed in patients with severe obstructive sleep apnea syndrome (OSAS).[16] Tárrega et al tried to exclude such patients by taking detailed medical histories and excluding subjects who might have OSAS, but OSAS can only be ruled out by full polysomnography.

In poor responders, nighttime P_{aCO_2} increase was associated with higher P_{aO_2} in the morning. P_{aO_2} at 7:00 AM was higher than at mid-day, awake, with the same rather low oxygen flow. Tárrega et al suggest that the P_{aO_2} increase was related to a higher fraction of inspired oxygen (F_{IO_2}), which was due to lower minute ventilation during sleep. One may also speculate that at the time of blood sampling the subjects might have been hyperventilating, which would have caused an immediate P_{aO_2} increase. However, that amount of time was insufficient to lower P_{aCO_2} because carbon dioxide accumulates in the body during sleep.

The nonhomogeneity of the studied group hinders interpretation of the data. Although all the subjects presented with airflow limitation, more than one third also had concomitant diseases such as tuberculosis, diaphragm paresis, or obesity. Those restrictive disorders may have had various effects on breathing pattern, pulmonary mechanics, and pulmonary gas exchange during sleep. Whatever were the mechanisms of blood gas disorders observed by Tárrega et al,[13] they were short-lasting. The next day at noon, on oxygen, P_{aCO_2} and pH had returned to the baseline values measured the day before while subjects were breathing air.

Based on their findings, Tárrega et al raise the question of the safety of increasing the oxygen flow during sleep to avoid nocturnal desaturation.[17] Theoretically, with a hypoxemic patient, increasing oxygen flow during sleep might further decrease the hypoxic drive to breathe, resulting in even higher P_{aCO_2}. Fortunately, that concern seems to be unwarranted. In the Nocturnal Oxygen Therapy Trial,[18] oxygen flow was increased by 1 L/min during sleep in all studied patients, and no adverse effects were reported. Śliwiński et al[19] investigated 17 stable COPD patients with respiratory failure (mean ± SD values for P_{aO_2} 53 ± 9 mm Hg and P_{aCO_2} 53 ± 9 mm Hg). S_{pO_2} and end-tidal carbon dioxide (measured via capnography) were measured continuously during 2 nights. During the first night patients breathed oxygen at 2 L/min, and during the other night at 3 L/min. The higher oxygen flow resulted in higher S_{pO_2}, with no increase in end-tidal carbon dioxide.

The effect of higher oxygen flow on P_{aCO_2} in hypercapnic patients remains to be studied. It seems, however, that hypoxemia during sleep is a more important clinical issue than oxygen-induced hypercapnia in a stable COPD patient.

Many COPD patients preserve satisfactory daytime oxygenation ($P_{aO_2} > 60$ mm Hg) while breathing air but suffer desaturation during sleep (nocturnal oxygen desaturation or NOD). Fletcher et al found NOD in 27% of 129 COPD patients who had daytime $P_{aO_2} > 70$ mm Hg and defined NOD as S_{pO_2} remaining below 90% for ≥ 5 minutes, with an S_{pO_2} nadir of ≤ 85%.[20]

Levi-Valensi et al[21] studied 40 COPD patients with daytime $P_{aO_2} > 60$ mm Hg and defined NOD as at least 30% of total sleep time with $S_{pO_2} < 90\%$. Almost half of the studied subjects desaturated during sleep.

Under many national guidelines for LTOT, NOD qualifies for nocturnal oxygen treatment.[22] In the United States, to qualify for nocturnal oxygen supplementation, overnight pulse oximetry must demonstrate $S_{pO_2} \leq 88\%$, regardless of the duration of desaturation. Nocturnal oxygen is also indicated if during-sleep P_{aO_2} decreases > 10 mm Hg or S_{pO_2} decreases > 5%, with signs or symptoms of hypoxemia (eg, cognitive disorders, restlessness, or insomnia).[23] However, the evidence supporting the necessity of nocturnal oxygen supplementation in NOD patients is rather contradictory.

Fletcher et al[24,25] reported that pulmonary artery catheter measurements of NOD patients revealed mild pulmonary hypertension (pulmonary artery pressure > 20 mm Hg) in the majority of studied subjects. Over 3 years of follow-up, NOD patients treated with nocturnal oxygen showed a slight but steady reduction in mean pulmonary artery pressure, averaging 3.7 mm Hg. NOD patients who received sham treatment (compressed air) showed pulmonary artery pressure increase of 3.9 mm Hg over the same period. Nondesaturators showed no change in pulmonary artery pressure.[26]

In a 3-year, retrospective, multi-center study, untreated patients with nocturnal hypoxemia appeared to have a higher risk of death.[27] It is interesting that 26% of patients without evidence of NOD at initial evaluation subsequently developed NOD (determined when re-studied 42 months later).[28]

Recently the rationale for nocturnal oxygen treatment was challenged by the European working group directed by Dr Weitzenblum in Strasbourg.[29] They investigated the effects of nocturnal desaturation on pulmonary hemodynamics in COPD patients with moderate hypoxemia during the day. Contrary to Fletcher et al, who found significantly higher mean pulmonary artery pressure in desaturators (23.3 ± 4.8 mm Hg vs 20.4 ± 4.2 mm Hg),[25] the Weitzenblum group showed no difference in pulmonary artery pressure between desaturators and nondesaturators, either at rest (19.4 ± 5.3 vs 18.7 ± 4.4 mm Hg) or during steady-state exercise of 40 watts (37.4 ± 8.7 vs 36.5 ± 8.8 mm Hg). These results seem to suggest that nocturnal desaturation does not induce clinically important pulmonary hypertension in COPD patients without or with only moderate daytime hypoxemia.[29]

After the initial investigation, patients who desaturated during sleep were randomly allocated to 2 groups. Forty-one patients commenced nocturnal oxygen supplementation, and 31 patients served as controls. The measured variables were pulmonary hemodynamics and survival.[30] Nocturnal oxygen therapy did not modify the evolution of pulmonary hemodynamics. In both groups the increase in pulmonary artery pressure after 2 years was negligible. Over a 3-year follow-up period, 9 patients from the treated group and 7 from the control group had died (a non-significant difference). The authors concluded that the prescription of nocturnal oxygen is probably not justified in COPD patients with isolated NOD. In the most recent study the Weitzenblum group confirmed that isolated nocturnal hypoxemia or sleep-related worsening of moderate daytime hypoxemia does not appear to favor the development of pulmonary hypertension.[31]

All COPD patients with NOD should have OSAS excluded. A crude assessment can be done by assessing over-

night S_{pO_2} readings. Among COPD patients, nocturnal desaturation episodes usually last approximately 20–30 minutes, whereas desaturations in OSAS patients last around 20–30 seconds. In patients with overlapping COPD and OSAS a combination of both features may be observed.

The treatment of all NOD COPD patients with nocturnal oxygen would result in a large number of subjects treated, at an enormous cost.[32] The rationale for the treatment of isolated NOD needs further study.

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REFERENCES

1. Dunne PJ. The demographics and economics of long-term oxygen therapy. Respir Care 2000;45(2):223–228; discussion 228–230.
2. O'Donohue WJ Jr, editor. Long-term oxygen therapy: scientific basis and clinical application. New York: Marcel Dekker; 1995.
3. Kacmarek RM. Delivery systems for long-term oxygen therapy. Respir Care 2000;45(1):84–92; discussion 92–94.
4. McNicholas WT. Impact of sleep in COPD. Chest 2000;117(2 Suppl): 48S–53S.
5. Robin ED. Some interactions between sleep and disease. Arch Intern Med 1958;102:669–675.
6. Koo KW, Sax DS, Snider GL. Arterial blood gases and pH during sleep in chronic obstructive pulmonary disease. Am J Med 1975; 58(5):663–670.
7. Flick MR, Block AJ. Continuous in vivo monitoring of arterial oxygenation in chronic obstructive lung disease. Ann Intern Med 1977; 86(6):725–730.
8. Douglas NJ, Calverley PMA, Leggett RJE, Brash HM, Flenley DC, Brezinova V. Transient hypoxaemia during sleep in chronic bronchitis and emphysema. Lancet 1979;1(8106):1–4.
9. Flenley DC. Clinical hypoxia: causes, consequences, and correction. Lancet 1978;1(8063):542–546.
10. Block AJ, Boysen PG, Wynne JW. The origins of cor pulmonale: a hypothesis. Chest 1979;75(2):109–110.
11. Śliwiński P, Łagosz M, Górecka D, Zieliński J. The adequacy of oxygenation in COPD patients undergoing long-term oxygen therapy assessed by pulse oximetry at home. Eur Respir J 1994;7(2):274–278.
12. Plywaczewski R, Śliwiński P, Nowiński A, Kamiński D, Zieliński J. Incidence of nocturnal desaturation while breathing oxygen in COPD patients undergoing long-term oxygen therapy. Chest 2000; 117(3):679–683.
13. Tarrega J, Güell R, Anton A, Mayos M, Farre À, Jerez FR, Sanchis J. Are daytime arterial blood gases a good reflection of nighttime gas exchange in patients on long-term oxygen therapy? Respir Care 2002; 47(8):882–886.
14. Rist KE, Doubenspeck JA, McGovern JF. Effects of non-REM sleep upon respiratory drive and the respiratory pump in humans. Respir Physiol 1986;63(2):241–256.
15. Radwan L, Dufmats H. Variations of arterial oxygen and carbon dioxide tension during 24 hours in chronic respiratory insufficiency. Scand J Respir Dis 1974;55(2):99–104.
16. Coccagna G, Mantovani M, Brignani F, Parchi C, Lugaresi E. Continuous recording of the pulmonary and systemic arterial pressure during sleep in syndromes of hypersomnia with periodic breathing. Bull Physiopathol Respir (Nancy)1972;8(5):1159–1172.
17. American Thoracic Society statement. Standards for the diagnosis and care of patients with chronic obstructive pulmonary disease. Am J Respir Crit Care Med 1995;152(5 Pt 2):S77–S121.
18. Nocturnal Oxygen Therapy Trial Group. Continuous or nocturnal oxygen therapy in hypoxemic chronic obstructive lung disease: a clinical trial. Ann Intern Med 1980;93(3):391–398.
19. Śliwiński P, Bieleń P, Nowiński A, Zieliński J. The effects of different oxygen flow rates on CO_2 retention in COPD patients qualified for LTOT (abstract). Eur Respir J 1999;14 Suppl 30:119s.
20. Fletcher EC, Miller J, Divine GW, Fletcher JG, Miller T. Nocturnal oxyhemoglobin desaturation in COPD patients with arterial oxygen tension above 60 mm Hg. Chest 1987;92(4):604–608.
21. Levi-Valensi P, Aubry P, Rida Z. Nocturnal hypoxemia and long-term therapy in COPD patients with daytime P_{aCO_2} 60–70 mmHg. Lung 1990;168 Suppl:770–775.
22. Zieliński J. Indications for long-term oxygen therapy: a reappraisal. Monaldi Arch Chest Dis 1999;54(2):178–182.
23. O'Donohue WJ Jr, Bowman TJ. Hypoxemia during sleep in patients with chronic obstructive pulmonary disease: significance, detection, and effects of therapy. Respir Care 2000;45(2):188–191; discussion 192–193.
24. Fletcher EC, Levin DC. Cardiopulmonary hemodynamics during sleep in subjects with chronic obstructive pulmonary disease: the effect of short- and long-term oxygen. Chest 1984;85(1):6–14.
25. Fletcher EC, Luckett RA, Miller T, Fletcher JG. Exercise hemodynamics and gas exchange in patients with chronic obstruction pulmonary disease, sleep desaturation, and a daytime P_{aCO_2} above 60 mm Hg. Am Rev Respir Dis 1989;140(5):1237–1245.
26. Fletcher EC, Luckett RA, Goodnight-White S, Miller CC, Qian W, Costarangos-Galarza C. A double-blind trial of nocturnal supplemental oxygen for sleep desaturation in patients with chronic obstructive pulmonary disease and a daytime P_{aCO_2} above 60 mm Hg. Am Rev Respir Dis 1992;145(5):1070–1076.
27. Fletcher EC, Donner CF, Midgren B, Zieliński J, Levi-Valensi P, Braghiroli A, et al. Survival in COPD patients with a daytime P_{aCO_2} greater than 60 mm Hg with and without nocturnal oxyhemoglobin desaturation. Chest 1992;101(3):649–655.
28. Fletcher EC, Scott D, Qian W, Luckett RA, Miller CC, Goodnight-White S. Evolution of nocturnal oxyhemoglobin desaturation in patients with chronic obstructive pulmonary disease and a daytime P_{aCO_2} above 60 mm Hg. Am Rev Respir Dis 1991;144(2):401–405.
29. Chaouat A, Weitzenblum E, Kessler R, Charpentier C, Ehrhart M, Levi Valensi P, et al. Sleep-related O_2 desaturation and daytime pulmonary haemodynamics in COPD patients with mild hypoxaemia. Eur Respir J 1997;10(8):1730–1735.
30. Chaouat A, Weitzenblum E, Kessler R, Charpentier C, Ehrhart M, Schott R, et al. A randomised trial of nocturnal oxygen therapy in chronic obstructive pulmonary disease patients. Eur Respir J 1999; 14(5):1002–1008.
31. Chaouat A, Weitzenblum E, Kessler R, Schott R, Charpentier C, Levi-Valensi P, et al. Outcome of COPD patients with mild daytime hypoxaemia with or without sleep-related oxygen desaturation. Eur Respir J 2001;17(5):848–855.
32. O'Donohue WJ Jr. Current concepts in supplemental oxygen therapy in patients with COPD. Semin Respir Crit Care Med 1999;20:341–350.

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New Technologies for Lighter Portable Oxygen Systems

Dr Tiep's report in this issue of Respiratory Care, which describes a new oxygen-conserving device, helps to draw attention to new trends in portable oxygen therapy.[1] As patients have moved from hospital to home, oxygen therapy has provided a cost-effective alternative for payers and an improved quality of life for patients. The Nocturnal Oxygen Therapy Trial[2] demonstrated the value of continuous oxygen therapy, and industry has provided systems to administer oxygen in the home. The Third Oxygen Consensus Conference concluded that most long-term oxygen therapy (LTOT) patients are ambulatory, and so portable oxygen should be available for patient mobility.[3]

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Initially, portable oxygen was available in small cylinders (Figure 1), but the operating times were not sufficient to meet patients' needs for longer times away from the stationary system. Larger (E-size) cylinders provided the volume for longer operating times but were too heavy to carry and not ambulatory, according to the Third Oxygen Consensus Conference.[3]

In 1965 the first liquid oxygen (LOX) system was introduced for home use. Union Carbide, working with Dr Tom Petty, developed a small portable system that had the weight and range to meet the patient's need for ambulation and that could be refilled from a base unit (Figure 2).[4] This started a major shift in home oxygen therapy, towards LOX as the first choice for mobile patients. In 1986 Medicare changed the way oxygen was paid for, moving from a fee-for-service program to a flat-rate, prospective payment method. Since LOX has a higher cost, associated with the service necessary to refill the base unit, home medical equipment providers moved to oxygen concentrators and small cylinders to control expenses.

The standard had been set, and patients and clinicians expected provision of a portable oxygen system with weight and range similar to a LOX portable. Oxygen-conserving devices (OCDs) thus became a viable alternative (Figure 3). OCDs had been available since 1984, but with a fee-for-service payment system there was no incentive for their use. Now an OCD can make a small cylinder last 3 times longer than was possible with continuous-flow oxygen and can compete with LOX portables.

Initially OCDs were not well received.[5] Many patients and clinicians said they tried the OCD, and it did not work. Some problems were related to technical issues of the sort

Fig. 1. The late Alvan Barach modeling a small high-pressure oxygen cylinder capable of being transfilled from a larger compressed gas cylinder. (From Reference 4.)

that occur with any new product on the market. Recent research provides another possible cause for poor results.[6] Each manufacturer determines what volume of gas to provide at each setting and reports that to be equivalent to continuous flow. Bench testing found that one device set on 4 was delivering 66 mL/breath, and another device set on 4 was delivering 34 mL/breath. That volume difference created confusion and the perception that the units did not work. With the focus on oxygen savings ratio rather than patient oxygenation, OCDs had an acceptance problem. Now it is recommended that the patient be tested with the prescribed OCD unit, at the expected activity levels.[7]

There is a lesson to be learned from this experience. Clinicians need to be informed of the performance capabilities of each piece of prescribed equipment. The respiratory prod-

Fig. 2. Patient transfilling a liquid oxygen portable system used in the home. (Courtesy of Thomas L Petty MD).

ucts industry is growing rapidly, and, without direction from clinicians, the manufacturers determine how a product should operate. In home care, marketing materials claim, for instance, that one oxygen device lasts longer than another or that the oxygen savings ratio is better than a competitor's. Those are important points if the patient is to be adequately oxygenated. If patient oxygenation is not the primary goal, you can conserve oxygen by reducing the flow from 2 L/min to 1 L/min and not have to purchase an OCD.

The LTOT market is growing rapidly, and patients are being placed on oxygen systems earlier in the course of disease.[8] The baby boom generation is moving into the years when many of them will become LTOT candidates. This younger patient group will be better informed of options for demand oxygen systems that meet their individual needs.

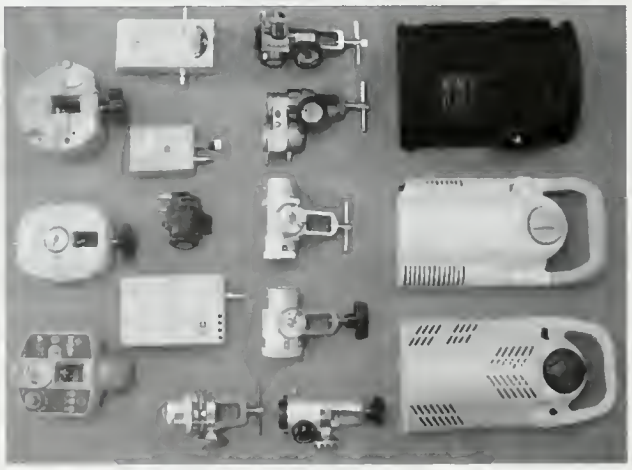

Fig. 3. Available oxygen conservation devices used with compressed gas or liquid oxygen. (Courtesy of Valley Inspired Products.)

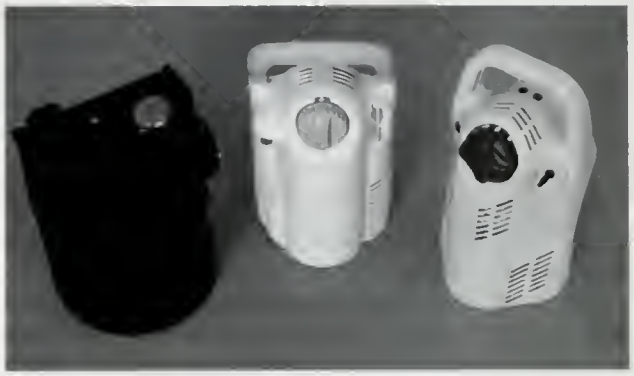

Fig. 4. Recently introduced liquid oxygen portables weighing between 3.5 and 4 lb. (Courtesy of Valley Inspired Products).

Recently-introduced products include lightweight, portable LOX systems that weigh less than 4 pounds and have an operating range of 6–8 hours at a typical setting of 2 (Figure 4). Concentrators that can fill cylinders in the home have been available for several years and are becoming good financial alternatives for providers of highly ambulatory patients (Figure 5). One manufacturer has received Food and Drug Administration clearance for a product that can liquefy oxygen from a concentrator and fill a LOX portable. Such home systems that can refill portable oxygen devices will give unrestricted freedom to patients who have in the past budgeted their oxygen supply based on the number of portable systems delivered by the provider.

Single-source systems that provide for both stationary and ambulatory needs will bring LTOT to a new level. A portable concentrator that has recently received Food and Drug Administration clearance for sale to the market weighs 9 pounds and can operate on battery for up to 50 minutes at a setting of 2 (Figure 6). This is the first step towards a single-source system, but this unit would not meet the recommendations of the Third Oxygen Consensus Conference for ambulatory oxygen.[3] New alternatives such as ceramic or electrochemical generation of oxygen may allow the development of oxygen systems that are light enough to carry and provide for the oxygen needs of highly ambulatory patients both at home and away.

Oxygen patients are becoming more involved in their therapy and are using the Internet to learn about their diseases and the therapy products available.[9] Quality of life is important to a patient, and knowing the options for care helps them to discuss their needs with the physician. Consumer-driven LTOT care is new to the industry and creates opportunities and challenges for clinicians. An informed patient will force the clinician to stay current on products and procedures.

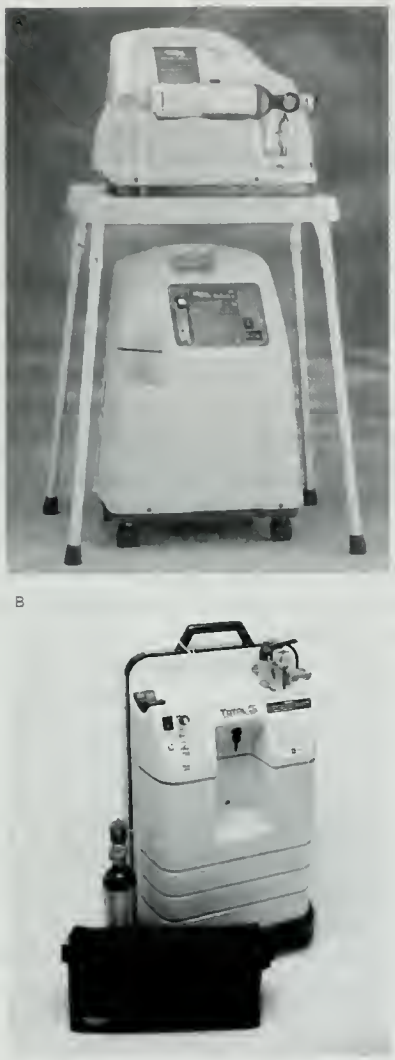

Fig. 5. Two commercially available concentrators that fill cylinders. A: Invacare HomeFill II. (Courtesy of Invacare Corporation.) B: Total O_2. (Courtesy of CHAD Therapeutics.)

The respiratory therapist needs to understand the operation of any piece of home-use respiratory equipment and to use that knowledge and his or her clinical skills to maximize the patient's benefit from that equipment. Each manufacturer will attempt to provide educational material on the operation, benefits, and value of its product, but the manufacturer's motive is to sell equipment. We have seen that claims of superior oxygen savings ratio or operating time were valuable for marketing but complicated the application of OCDs. With the rapid development of new LTOT products, the clinician needs to be involved and informed of the clinical efficacy of the products and their potential value to the patients.

Fig. 6. A battery-operated oxygen concentrator. This device weighs 9 lb and the batteries last a little less than an hour. (Courtesy of AirSep Corporation.)

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REFERENCES

1. Tiep BL, Barnett J, Schiffman G, Sanchez O, Carter R. Maintaining oxygenation via demand oxygen delivery during rest and exercise. Respir Care 2001;47(8):887–892.
2. Continuous or nocturnal oxygen therapy in hypoxemic chronic obstructive lung disease: a clinical trial. Nocturnal Oxygen Therapy Trial Group. Ann Intern Med 1980;93(3):391–398.
3. Conference Report. New problems in supply, reimbursement, and certification of medical necessity for LTOT (Summary of the third consensus conference held in Washington, DC, Mar 15–16, 1990). Am Rev Respir Dis 1990;142:721–724.
4. Petty TL. Historical highlights of long-term oxygen therapy. Respir Care 2000;45(1):29–36; discussion 36–38.
5. Block AJ. Intermittent flow oxygen devices: technically feasible, but rarely used (editorial). Chest 1984;86(5):657–658.
6. Bliss PL, McCoy RW, Adams AB. A bench study comparison of demand oxygen delivery systems and continuous flow oxygen. Respir Care 1999;44(8):925–931.
7. Petty TL, Casaburi R. Recommendations of the Fifth Oxygen Consensus Conference. Respir Care 2000;45(8):957–961.
8. Frost and Sullivan. United States Oxygen Therapy Devices Market. 2001. Available at http://www.frost.com (accessed June 6, 2002).
9. Emphysema Foundation For Our Right To Survive (EFFORTS). http://www.emphysema.net (accessed June 6, 2002).

Are Daytime Arterial Blood Gases a Good Reflection of Nighttime Gas Exchange in Patients on Long-Term Oxygen Therapy?

Julia Tárrega MD, Rosa Güell PhD, Antonio Antón MD, Merce Mayos PhD, Ariadna Farré RN, Francisco R Jerez MD, and Joaquín Sanchis PhD

OBJECTIVE: Compare nighttime and daytime arterial blood gas values in patients undergoing long-term oxygen therapy (LTOT). METHODS: We studied 39 LTOT patients with chronic airflow limitation. Oxygen from an oxygen concentrator was administered via nasal prongs until daytime blood oxygen saturation (measured via pulse oximetry [S_{pO_2}]) was ≥ 90%. Arterial blood samples were drawn at 6:00 PM, while the subject breathed room air, and also during oxygen administration at night (3:00 AM), early in the morning (7:00 AM), and at noon. S_{pO_2} was measured throughout the night. RESULTS: Mean patient age was 70 ± 7 yr. All patients suffered severe chronic airflow limitation (mean forced expiratory volume in the first second 28 ± 9% of predicted). The mean oxygen flow administered was 1.41 ± 0.6 L/min. Mean overnight S_{pO_2} was 92 ± 2.5%, with 21.5 ± 28% of recording time under 90%. There were statistically significant differences between P_{aO_2}, P_{aCO_2}, and pH obtained at 3:00 AM and noon and between 7:00 AM and noon, while the patients breathed the same oxygen concentration. The differences between the 3:00 and 7:00 AM values were not significant. In 23 patients (59%) we observed a P_{aCO_2} increase > 10 mm Hg and/or a pH decrease to < 7.33 during that period, indicating poor response to LTOT. CONCLUSIONS: Daytime arterial blood gas measurements do not reflect nighttime gas exchange. However, samples taken early in the morning (7:00 AM) do seem to reflect arterial blood gases during the night and can therefore be used for setting and monitoring nighttime oxygen flow. *Key words: hypercapnia, COPD, long-term oxygen therapy, LTOT, nocturnal hypoventilation, ABG, arterial blood gas measurement.* [Respir Care 2002;47(8):882–886]

Introduction

Long-term oxygen therapy (LTOT) improves measures of pulmonary hemodynamics in patients with chronic obstructive pulmonary disease (COPD) and lengthens the survival of patients with both COPD and chronic respiratory failure.[1,2] It is generally believed that oxygen can provide the same benefits to patients with respiratory failure due to diseases other than COPD, such as bronchiectasis, pulmonary fibrosis, cystic fibrosis, or tuberculosis sequelae, although well-designed studies that support that hypothesis are lacking.[3]

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According to widely accepted guidelines,[4–6] oxygen flow to correct hypoxemia is titrated based on daytime arterial blood gas (ABG) values or oxyhemoglobin saturation. A nighttime oxygen flow increase of 1 L/min more than the daytime flow is recommended to avoid nighttime desaturation.[4–7] However, substantial changes in ventilation and gas exchange take place during sleep.[8] Oxygen therapy mitigates nocturnal desaturation,[9–11] but studies of transcutaneously measured P_{CO_2} suggest that hypoventilation and consequent hypercapnia may develop.[10–14] Such re-

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ports suggest that current methods for titrating nighttime oxygen flow might not be optimal, although studies of nighttime ABG values in these patients are lacking. Recently, in a small sample of sleeping patients receiving LTOT for severe airflow obstruction due to cystic fibrosis, Milross et al[15] measured ABG values and found that nighttime P_{aCO_2} increased significantly and pH decreased.

Our hypothesis for the present study was that a considerable proportion of patients receiving oxygen therapy develop substantial hypercapnia and respiratory acidosis during sleep, even though daytime ABG values appear to indicate that oxygen flow has been appropriately titrated. To test this hypothesis we compared nighttime and daytime ABG values in patients undergoing oxygen therapy.

Methods

From November 1997 to December 1998 we prospectively enrolled 39 patients with chronic respiratory failure associated with chronic airflow limitation (forced expiratory volume in the first second [FEV_1] < 80% of predicted and ratio of FEV_1 to forced vital capacity [FVC] < 70%).[4,5] All patients fulfilled conventional criteria for LTOT.[4–6] Each had been clinically stable for at least 4 weeks before the study. Patients and their partners were asked about the presence of symptoms of obstructive sleep apnea syndrome (OSAS): daytime sleepiness, snoring, and apnea during sleep. Patients with symptoms suggestive of OSAS were excluded. All participants had undergone pulmonary function testing, including spirometry and determination of static pulmonary volumes and diffusing capacity of the lung for carbon monoxide. Each participant was in the hospital for 1 night for the study. The ABG samples were obtained via radial artery catheter (Vigon, Écouen, France). Oxygen from a concentrator (Zefir, Air Liquide/Taema, Paris [Antony], France) was administered via nasal prongs. According to guidelines,[4–6] oxygen flow was set to keep daytime $S_{pO_2} \geq 90\%$ at rest, and, once titrated, flow was not subsequently modified throughout the study period. ABG values were obtained at baseline (on room air) at around 6:00 PM, and, after adjusting the oxygen flow, at night (3:00 AM), early in the morning (7:00 AM), and at noon. All arterial blood samples were obtained with the patient supine. Oxygen saturation was also recorded overnight via pulse oximetry (PULSOX-5, Minolta, Anandic Medical Systems, Diessenhofer, Switzerland).

We judged that a patient responded poorly to oxygen therapy if P_{aCO_2} increased > 10 mm Hg and/or pH decreased to < 7.33 in any of the ABG samples.

Results are presented as mean ± standard deviation. One-way analysis of variance and the 2-tailed paired *t* test were used to determine the statistical differences between the ABG samples. A p value of < 0.05 was considered significant.

Results

Table 1 shows demographic and pulmonary function characteristics of the participants. All 39 subjects had severe chronic air flow limitation, and 25 had COPD. The other diagnoses included tuberculosis sequelae (6), bronchiectasis (5), diaphragmatic paresis (1), kyphoscoliosis (1), and obesity associated with chronic air flow limitation (1). Seventeen patients were already on LTOT at the time of enrollment and 22 were prescribed domiciliary oxygen therapy according to standard criteria[4–6] for the first time as they entered the study. The mean oxygen flow administered was 1.41 ± 0.6 L/min.

Table 1. Clinical Characteristics and Results of Pulmonary Function Testing

	Total $n = 39$	COPD $n = 25$	CAFL in Non-COPD Patients $n = 14$
Sex (m/f)	36/3	23/2	13/1
Age (y)	70 ± 7	70 ± 7	69 ± 6
BMI (kg/m^2)	29 ± 4	29 ± 5	29 ± 4
FEV_1 (% of predicted)	28 ± 9	28 ± 9	28 ± 11
FEV_1/FVC (%)	39 ± 12	35 ± 9	45 ± 17
RV (% of predicted)	153 ± 64	167 ± 69	124 ± 44
TLC (% of predicted)	96 ± 26	104 ± 24	80 ± 25
D_{LCO} (% of predicted)	46 ± 15	48 ± 15	39 ± 13
Baseline P_{aO_2} (mm Hg)	54 ± 8	55 ± 7	53 ± 9
Baseline P_{aCO_2} (mm Hg)	54 ± 7	53 ± 4	54 ± 10
Baseline pH	7.39 ± 0.03	7.39 ± 0.002	7.39 ± 0.003

COPD = chronic obstructive pulmonary disease
CAFL = chronic airflow limitation
BMI = body-mass index
FEV_1 = forced expiratory volume in the first second
FVC = forced vital capacity
RV = residual volume
TLC = total lung capacity
D_{LCO} = diffusing capacity of the lung for carbon monoxide
Values are mean ± SD

Table 2 shows the ABG values. There were no significant differences in the P_{aO_2}, P_{aCO_2}, or pH values between

Table 2. Arterial Blood Gas Values

	3:00 AM	7:00 AM	Noon
P_{aO_2} (mm Hg)	82 ± 12	80 ± 12	67 ± 10*
P_{aCO_2} (mm Hg)	60 ± 10	61 ± 11	55 ± 8*
pH	7.34 ± 0.04	7.34 ± 0.04	7.38 ± 0.03*

Values are mean ± SD
*Differences in P_{aO_2}, P_{aCO_2}, and pH were significant between the noon value and both the 3:00 AM and 7:00 AM values ($p < 0.05$)

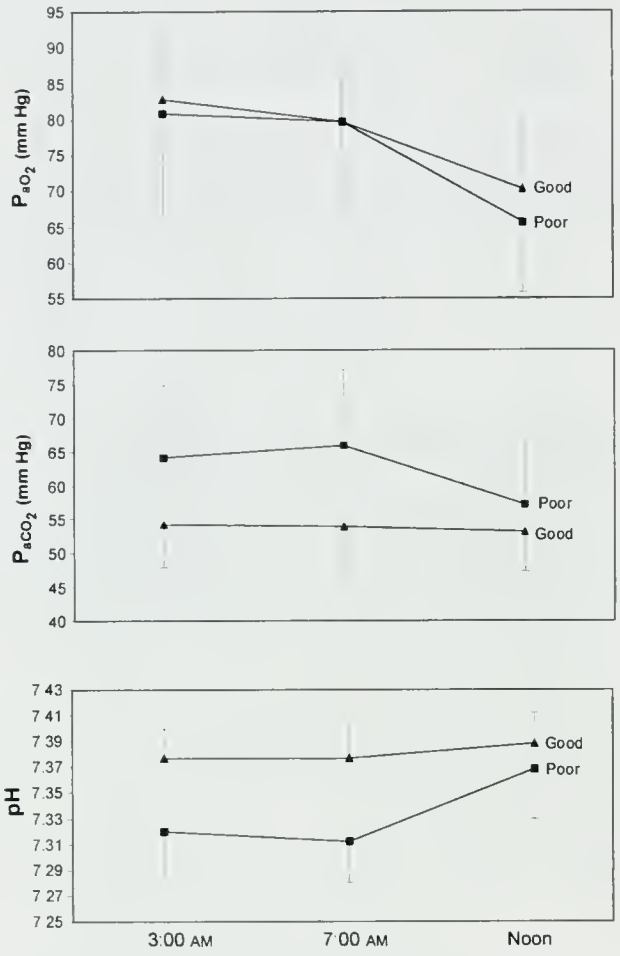

Fig. 1. Arterial blood gas values during oxygen therapy, at 3:00 AM, 7:00 AM, and noon, for poor responders (square data points) and good responders (triangle data points). Error bars represent 2 standard deviations.

the samples taken at 3:00 AM and those taken at 7:00 AM. However, the differences were significant between the samples taken at 3:00 AM and noon and between those taken at 7:00 AM and noon. Thus, at night and early in the morning, P_{aCO_2} was significantly higher and pH was significantly lower.

The 23 patients (59%) who were poor responders had a mean P_{aCO_2} of 65 mm Hg and a mean pH of 7.31. Figure 1 shows the means and 2 standard deviations of the ABG values. Sixty percent of the COPD patients and 57% of the non-COPD patients were poor responders. We found no significant differences between the poor responders COPD patients and poor responders non-COPD patients (Table 3).

The mean overnight oxygen saturation for all patients was 92 ± 2.5%. The percentage of recording time during which S_{pO_2} was < 90% was 21.5 ± 28%. Using "S_{pO_2} < 90% < 30% of the time" as the cutoff for "adequate oxygenation,"[16] we found that 30% of our participants were not assured satisfactory nighttime oxygenation when flow was set according to daytime blood gas values.

Discussion

Our study is the first to demonstrate that a considerable percentage of both COPD and non-COPD patients undergoing LTOT based on conventional criteria develop severe hypercapnia and respiratory acidosis during sleep and that blood gas values obtained while awake do not reflect their during-sleep hypercapnia. Our study also revealed, on the other hand, that the early morning (7:00 AM) ABG values did reflect nighttime blood gas values.

During sleep, ventilation decreases substantially in patients with chronic respiratory diseases, potentially causing dangerous gas exchange abnormalities. These changes have been attributed to the worsening of ventilation-perfusion relationships[17] and to hypoventilation during sleep, mainly due to reduced tidal volume (V_T). The mechanisms include increased upper airway resistance,[17] decreased neural drive,[18] and hypotonia of respiratory muscles.[17,19,20] This situation may worsen with oxygen therapy because hypoxic drive is decreased[21] and/or the abnormality of ventilation-perfusion relationships increases when hypoxic pulmonary vasoconstriction is amended.[22,23] In COPD patients the ventilatory response to carbon dioxide decreases[24] during sleep, and shallow breathing and decreased V_T develop, which increases the ratio of dead space to V_T and tends to worsen hypercapnia.[14,25–28]

International guidelines,[4–6] supported by recent studies,[7] recommend increasing the nighttime oxygen flow by 1 L/min, but our findings indicate that that recommendation should be followed with caution. We observed a high percentage (59%) of patients with substantial hypercapnia and respiratory acidosis associated with nighttime oxygen therapy. Given that 30% of our patients were not assured adequate S_{pO_2}, we hypothesize that nighttime respiratory acidosis might worsen even further for such patients if current recommendations are followed. However, the long-term clinical importance of worsening nighttime hypercapnia and acidosis is unclear. Nighttime hypercapnia has been reported to increase arousal frequency and thus may be related to poor sleep quality,[29,30] affecting the course of disease and the quality of life for these patients.[31]

The finding that nighttime P_{aO_2} was higher than during the day was unexpected. Taking into account the changes in ventilation that COPD patients suffer during sleep and the fact that oxygen therapy was administered via nasal prongs, a possible explanation for that observation is that during sleep the patient might receive a higher fraction of inspired oxygen because of lower V_T.

Among the 59% of patients who were poor responders, some might have had undiagnosed OSAS. Goldstein et al[10] studied oxygen saturation response and transcutane-

Table 3. Data from Chronic Airflow Limitation Patients With and Without Chronic Obstructive Pulmonary Disease

	Baseline	3:00 AM	7:00 AM	Noon	Baseline	3:00 AM	7:00 AM	Noon
	With Chronic Obstructive Pulmonary Disease							
	Poor Responders* (n = 15)				Good Responders (n = 10)			
P_{aO_2} (mm Hg)	53 ± 8	79 ± 13	77 ± 13	65 ± 8	56 ± 5	84 ± 10	79 ± 9	71 ± 9
P_{aCO_2} (mm Hg)	54 ± 5	63 ± 11	63 ± 10	55 ± 7	51 ± 4	56 ± 5	55 ± 5	54 ± 3
pH	7.38 ± 0.018	7.32 ± 0.003	7.31 ± 0.001	7.36 ± 0.001	7.41 ± .018	7.36 ± 0.0004	7.36 ± 0.0003	7.38 ± 0.047
BMI (kg/m^2)	29 ± 5				30 ± 5			
FEV_1 (% of predicted)	27 ± 9				29 ± 10			
FEV_1/FVC (%)	35 ± 7				36 ± 10			
TLC (% of predicted)	97 ± 24				108 ± 16			
RV (% of predicted)	156 ± 72				170 ± 58			
	Without Chronic Obstructive Pulmonary Disease							
	Poor Responders (n = 8)				Good Responders (n = 6)			
P_{aO_2} (mm Hg)	50 ± 10	83 ± 18	83 ± 21	65 ± 13	56 ± 7	76 ± 6	77 ± 2	66 ± 3
P_{aCO_2} (mm Hg)	58 ± 10	68 ± 11	72 ± 12	61 ± 13	50 ± 8	51 ± 6	50 ± 6	48 ± 6
pH	7.37 ± 0.0007	7.30 ± 0.043	7.30 ± 0.036	7.36 ± 0.05	7.42 ± 0.00005	7.39 ± 0.002	7.39 ± 0.027	7.40 ± 0.02
BMI (kg/m^2)	30 ± 3				28 ± 5			
FEV_1 (% of predicted)	27 ± 10				30 ± 12			
FEV_1/FVC (%)	51 ± 20				39 ± 11			
TLC (% of predicted)	74 ± 27				86 ± 23			
RV (% of predicted)	106 ± 31				141 ± 51			

*Poor responder = P_{aCO_2} increased > 10 mm Hg and/or pH decreased to < 7.33
BMI = body mass index
FEV_1 = forced expiratory volume in the first second
FVC = forced vital capacity
TLC = total lung capacity
RV = residual volume

ously measured P_{CO_2} during sleep with 15 clinically stable COPD patients with hypercapnic respiratory failure and receiving oxygen. P_{CO_2} increased by only 6 mm Hg. Only in 3 of their patients, who also presented with associated OSAS, was the P_{CO_2} increase > 10 mm Hg.

Our study was carried out under the usual conditions of clinical practice, with patients receiving LTOT based on conventional criteria. Thus, we excluded those who reported symptoms suggestive of OSAS, but did not screen for OSAS by polysomnography, as such unconventional exclusion might have introduced bias by creating a study sample that would not have been typical of those for whom LTOT is conventionally recommended. Polysomnographic study of patients with nighttime hypercapnic response to oxygen therapy would be useful to determine whether OSAS plays an important role and to help guide alternative therapies.

Conclusions

Daytime ABG values do not reflect nighttime gas exchange, as nighttime hypercapnia seems to develop in over half of LTOT patients. On the other hand, ABG values measured early in the morning do closely reflect nighttime oxygenation, and we therefore recommend early-morning measurements for setting and monitoring nighttime oxygen therapy.

REFERENCES

1. Nocturnal Oxygen Therapy Trial Group. Continuous or nocturnal oxygen therapy in hypoxemic chronic obstructive lung disease: a clinical trial. Ann Intern Med 1980;93(3):391–398.
2. Report of the Medical Research Council Working Party. Long term domiciliary oxygen therapy in chronic hypoxic cor pulmonale complicating chronic bronchitis and emphysema. Lancet 1981;1(8222): 681–686.
3. Weiss SM, Pierson DJ. Long-term oxygen therapy in disorders other than COPD. In: O'Donohue WJ. Long-term oxygen therapy. Scientific basis and clinical application. Lung Biology in Health and Disease. Vol 81. New York: Marcel Dekker; 1995:101–116.
4. Standards for the diagnosis and care of patients with chronic obstructive pulmonary disease. ATS Statement. Am J Respir Crit Care Med 1995;152(5 Pt 2):S77–S121.
5. Siafakas NM, Vermeire P, Pride NB, Paoletti P, Gibson J, Howard P, et al. Optimal assessment and management of chronic obstructive

pulmonary disease (COPD). The European Respiratory Society Task Force. Eur Respir J 1995;8(8):1398–1420.
6. Sanchez Agudo L, Cornudella R, Estopa Miro R, Molinos Martin L, Servera Pieras E. [Guidelines for indications and use of domiciliary continuous oxygen (DCO) therapy. SEPAR guidelines] Arch Bronconeumol 1998;34(2):87–94. *article in Spanish*
7. Plywaczewski R, Sliwinski P, Nowinski A, Kaminski D, Zielinski J. Incidence of nocturnal desaturation while breathing oxygen in COPD patients undergoing long-term oxygen therapy. Chest 2000; 117(3):679–683.
8. Douglas NJ. Control of ventilation during sleep. Clin Chest Med 1985;6(4):563–575.
9. Morrison D, Skwarski KM, MacNee W. The adequacy of oxygenation in patients with hypoxic chronic obstructive pulmonary disease treated with long-term domiciliary oxygen. Respir Med 1997;91(5): 287–291.
10. Goldstein RS, Ramcharan V, Bowes G, McNicholas WT, Bradley D, Phillipson E. Effects of supplemental nocturnal oxygen on gas exchange in patients with severe obstructive lung disease. N Engl J Med 1984;310(7):425–429.
11. Fletcher EC, Levin DC. Cardiopulmonary hemodynamics during sleep in subjects with chronic obstructive pulmonary disease: the effect of short- and long-term oxygen. Chest 1984;85(1):6–14.
12. Leitch AG, Clancy LJ, Leggett RJE, Tweeddale P, Dawson P, Evans JI. Arterial blood gas tensions, hydrogen ion, and electroencephalogram during sleep in patients with chronic ventilatory failure. Thorax 1976;31(6):730–735.
13. Meecham Jones JD, Paul EA, Jones PW, Wedzicha JA. Nasal pressure support ventilation plus oxygen compared with oxygen therapy alone in hypercapnic COPD. Am J Respir Crit Care Med 1995; 152(2):538–544.
14. Griffiths TL, Fernando SSD, Saunders KB. Effect of adenosine infusion on oxygen induced carbon dioxide retention in severe chronic obstructive pulmonary disease. Thorax 1996;51(11):1083–1086.
15. Milross MA, Piper AJ, Norman M, Becker HF, Willson GN, Grunstein RR, et al. Low-flow oxygen and bilevel ventilatory support: effects on ventilation during sleep in cystic fibrosis. Am J Respir Crit Care Med 2001;163(1):129–134.
16. Levi-Valensi P, Weitzenblum E, Rida Z, Aubry P, Braghiroli A, Donner C, et al. Sleep-related oxygen desaturation and daytime pulmonary haemodynamics in COPD patients. Eur Respir J 1992;5(3): 301–307.
17. Becker HF, Piper AJ, Flynn WE, McNamara SG, Grunstein RR, Peter JH, Sullivan CE. Breathing during sleep in patients with nocturnal desaturation. Am J Respir Crit Care Med 1999;159(1):112–118.
18. Ballard RD, Clover CW, Suh BY. Influence of sleep on respiratory function in emphysema. Am J Respir Crit Care Med 1995;151(4): 945–951.
19. Heijdra YF, Dekhuijzen PNR, van Herwaarden CLA, Folgering HTM. Nocturnal saturation and respiratory muscle function in patients with chronic obstructive pulmonary disease. Thorax 1995;50(6):610–612.
20. McNicholas WT. Impact of sleep in respiratory failure. Eur Respir J 1997;10(4):920–933.
21. Hanson CW 3rd, Marshall BE, Frasch HF, Marshall C. Causes of hypercarbia with oxygen therapy in patients with chronic obstructive pulmonary disease. Crit Care Med 1996;24(1):23–28.
22. Robinson TD, Freiberg DB, Regnis JA, Young IH. The role of hypoventilation and ventilation-perfusion redistribution in oxygen-induced hypercapnia during acute exacerbations of chronic obstructive pulmonary disease. Am J Respir Crit Care Med 2000;161(5): 1524–1529.
23. Benditt JO. Adverse effects of low-flow oxygen therapy. Respir Care 2000;45(1):54–61; discussion 61–64.
24. Gelb A, Klein E, Schiffman P, Lugliani R, Aronstam P. Ventilatory response and drive in acute and chronic obstructive pulmonary disease. Am Rev Respir Dis 1977;116(1):9–16.
25. Dunn WF, Nelson SB, Hubmayr RD. Oxygen-induced hypercarbia in obstructive pulmonary disease. Am Rev Respir Dis 1991;144(3 Pt 2):526–530.
26. Aubier M, Murciano D, Fournier M, Milic-Emili J, Pariente R, Derenne JP. Central respiratory drive in acute respiratory failure of patients with chronic obstructive pulmonary disease. Am Rev Respir Dis 1980;122(2):191–199.
27. Aubier M, Murciano D, Milic-Emili J, Touaty E, Daghfous J, Pariente R, Derenne JP. Effects of the administration of O_2 on ventilation and blood gases in patients with chronic obstructive pulmonary disease during acute respiratory failure. Am Rev Respir Dis 1980; 122(5):747–754.
28. Sassoon C, Hassell KT, Mahutte CK. Hyperoxic-induced hypercapnia in stable chronic obstructive pulmonary disease. Am Rev Respir Dis 1987;135(4):907–911.
29. Fleetham J, West P, Mezon B, Conway W, Roth T, Kryger M. Sleep, arousals, and oxygen desaturation in chronic obstructive pulmonary disease: the effect of oxygen therapy. Am Rev Respir Dis 1982; 126(3):429–433.
30. McKeon JL, Murree-Allen K, Saunders NA. Supplemental oxygen and quality of sleep in patients with chronic obstructive lung disease. Thorax 1989;44(3):184–188.
31. Criner GJ. Effects of long-term oxygen therapy on mortality and morbidity. Respir Care 2000;45(1):105–118.

Maintaining Oxygenation via Demand Oxygen Delivery During Rest and Exercise

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BACKGROUND: Oxygen-conserving devices have been the foundation of highly portable oxygen systems that enable hypoxemic chronic lung disease patients to live active lives. Pulsing demand oxygen delivery systems (DODS) can adequately oxygenate most patients at rest and usually during exercise. However, some patients desaturate during exercise at DODS settings equivalent to continuous-flow oxygen. OBJECTIVE: Determine if design enhancements of the Oxymatic 401 DODS (including increased sensitivity, earlier inspiratory delivery, larger pulse volume, modified pulse contour, and higher settings) better maintain blood oxygen saturation (measured via pulse oximetry [S_{pO_2}]) in patients who severely desaturate during exercise. METHODS: We compared patients receiving DODS oxygen and patients receiving continuous-flow oxygen, during both rest and iso-exercise. SETTING: This study was conducted in the Pulmonary Rehabilitation Program at Mission Hospital, Regional Medical Center, Mission Viejo, California. SUBJECTS: We studied 10 patients with chronic lung disease and difficulty maintaining exercise S_{pO_2} with DODS. Nine patients had chronic obstructive pulmonary disease and one had restrictive lung disease. The group's (mean ± SD) demographic and physiologic values included: age 67 ± 7 y, forced expiratory volume in the first second 0.79 ± 0.3 L, forced vital capacity 1.7 ± 0.7 L, and S_{pO_2} 88%. INTERVENTIONS: Resting S_{pO_2} was measured during continuous oxygen flow and during DODS, at identical settings. S_{pO_2} was allowed to stabilize at each level before recording. During treadmill exercise at identical work loads we measured continuous flow and DODS S_{pO_2} at the same settings. If the DODS S_{pO_2} was less than during the equivalent continuous flow, the DODS setting was increased for another treadmill trial. RESULTS: At rest the DODS was equivalent to continuous flow: S_{pO_2} was 93.7 ± 2.1% with DODS and 93.8 ± 1.9% with continuous flow. During exercise at corresponding settings DODS S_{pO_2} was 90.5 ± 3.8% and continuous-flow S_{pO_2} was 93.1 ± 3.1%. Six subjects had S_{pO_2} > 90%, with 4 of them achieving equivalency. At higher DODS settings S_{pO_2} became equivalent: 92.5 ± 2.8%. The DODS oxygen-use efficiency advantage was 6.7-fold at rest and 5.6-fold during exercise, except with subjects who required a higher exercise DODS setting, with whom the DODS advantage was 4.3-fold. CONCLUSIONS: The redesigned Oxymatic 401 DODS maintains adequate S_{pO_2} during rest and exercise, but some patients require the higher delivery settings. We recommend that all patients prescribed DODS undergo exercise evaluation with the prescribed DODS to ensure efficacy and determine the DODS settings required to maintain S_{pO_2} at the prescribed limits. *Key words: oxygen delivery, oxygen conservation, exercise, treadmill, oxygen saturation.* [Respir Care 2002;47(8):887–892]

Introduction

The primary goals of medical care for patients with chronic lung disease are to manage the disease, improve physical and psychological function, and enhance quality of life. Oxygen therapy for patients with chronic obstructive pulmonary disease and hypoxemia is directed toward maintaining oxygen saturation at rest and preventing desaturation during exercise and sleep. Several portable oxygen systems have been designed to provide adequate oxygenation while minimizing the size and weight of the

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This research was conducted at the Pulmonary Rehabilitation Program at Mission Hospital Regional Medical Center, Mission Viejo, California, and was supported in part by CHAD Therapeutics Inc, Chatsworth, California.

oxygen system. Further benefit is achieved through the use of portable systems that promote ambulation away from the home.

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Portable oxygen supports chronic lung disease patients' ability to maintain an active lifestyle and participate in many of life's pleasures, thus improving quality of life. Oxygen conservation technology is among the most important developments in portable oxygen therapy in recent years. As the efficiency of oxygen delivery devices improve, these devices become smaller and more portable, thereby reducing the burden on the patient, from both an economic and physical perspective. A variety of oxygen-conserving devices, including reservoir cannulas,[1,2] transtracheal catheters,[3,4] and demand oxygen delivery systems (DODS),[5–7] have provided the hypoxemic patient with choices and tradeoffs. The most efficient of these systems is the DODS, which delivers oxygen early in the inhalation phase, ensuring that a bolus of oxygen reaches the alveoli and avoiding the waste of oxygen that occurs with systems that deliver oxygen throughout inhalation and exhalation. DODS oxygen-use efficiency ranges from 2-fold to 7-fold better than continuous-flow oxygen delivery.[8] That is, DODS can achieve the same oxygen saturation levels as continuous flow while using only one half to one seventh the oxygen.

Previous studies found DODS effective during rest, sleep, and exercise.[1,7–9] Recently, however, it became apparent that some DODS users desaturate during exercise at DODS settings that correspond to continuous flow. In other words, DODS settings equivalent to continuous flow were insufficient to achieve equivalent oxygen saturation. That difference in equivalency between rest and exercise appears to result from several physiologic and mechanical factors. In an attempt to improve on several of those factors and fully meet the oxygenation needs of patients who do not adequately saturate with existing DODS during exercise, one of the DODS models, the Oxymatic 401 (CHAD Therapeutics, Chatsworth, California), was redesigned. Specifically, the pulse volume and sensitivity to inspiratory flow were increased, the delivery waveform was modified, and higher flow settings were added, all while retaining the DODS oxygen-use efficiency advantage.

The present study was designed to investigate if those design modifications achieve adequate oxygenation in DODS patients who desaturate during exercise.

Methods

Subjects

Ten patients were recruited from Mission Hospital Regional Medical Center's Pulmonary Rehabilitation Program. Per institutional guidelines, each participant had the study explained in detail prior to signing the consent form. All the participants had histories of severe chronic lung disease. The group's demographic and physiologic (mean ± SD) values included: age 67 ± 7 yr, forced expiratory volume in the first second 0.79 ± 0.3 L, forced vital capacity 1.7 ± 0.7 L, and blood oxygen saturation (on room air, measured via pulse oximetry [S_{pO_2}]) 88 ± 0.3%. All the participants regularly required supplemental oxygen, particularly during exercise, and all were hypoxemic, particularly during mild exertion. Most participants had histories of desaturating during exercise while using various DODS with DODS settings equivalent to continuous flow. They were stable in their diseases, with no signs of exacerbation at the time of testing. Nine of the patients had chronic obstructive pulmonary disease. One patient had restrictive lung disease due to severe kyphoscoliosis. All the patients were able to walk on a treadmill for at least 2 minutes, for multiple sessions, at a low work load.

Equipment

The DODS tested was the Oxymatic 401. This device, like previous models, was designed to deliver oxygen only early in the inhalation phase and to not deliver oxygen during exhalation and dead-space inhalation (ie, late in the inhalation phase). This approach maximizes oxygen gas exchange in the lung and takes into account several basic physiologic principles with respect to alveolar gas exchange and ventilation. Design and engineering enhancements included:

1. The oxygen pulse bolus was increased from 35 mL to 40 mL of 100% oxygen. The delivery begins within milliseconds of the beginning of inhalation, thus ensuring participation in alveolar gas exchange.

2. New settings (5 and 6) were added; they correspond to 5 and 6 L/min, delivering pulse volumes of 50 and 60 mL, respectively. This enhancement was necessary to maintain saturation by delivering a greater quantity of oxygen

Brian L Tiep MD presented a version of this report at the International Conference of the American Thoracic Society, April 23–28, 1999.

Brian L Tiep MD is the designer of and receives royalties for several oxygen delivery devices, including the one studied for this report. Every effort was made to perform an unbiased study, and investigators with no financial interest carefully monitored the study. Oscar Sanchez is a design engineer at CHAD Therapeutics Inc, and he was involved with the equipment portion of the study with Dr Tiep.

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while maintaining the early-inhalation delivery principle and, thus, overall oxygen savings.

3. The pulse waveform was adjusted to better match the patient's inspiratory breathing pattern. Like the earlier model, the redesigned Oxymatic 401 delivers oxygen pulses on an intermittent breath delivery schedule in Settings 1, 2, and 3 (Setting 1: delivery during 1 out of 4 every breaths; Setting 2: delivery during 2 out of 4 breaths; Setting 3: delivery during 3 out of 4 breaths). On Settings 4, 5, and 6, the Oxymatic 401 delivers oxygen during every breath.

Protocol

All subjects had oxygen prescriptions previously written by their physicians, for both rest and exercise, intended to maintain S_{pO_2} between 92% and 94%. Room-air S_{pO_2} was recorded from all participants, at rest, for 10 min, using a calibrated pulse oximeter (Nonin, Plymouth, Minnesota). Continuous-flow oxygen was adjusted, at rest, to the prescribed setting and S_{pO_2} was recorded when the oximeter signal stabilized. The patient was then switched to the DODS at the corresponding flow setting and, again, S_{pO_2} was recorded when the oximeter signal stabilized.

Each patient underwent at least 2 treadmill exercise trials at identical submaximal work loads (typically 1.5 mi/h, 0% grade, for 2 min) with the DODS suspended from the treadmill rail so as not to add additional weight to the patient during the exercise. The first treadmill trial was performed while the patient received continuous-flow oxygen adjusted to the prescribed exercise setting. S_{pO_2} was recorded at the end of exercise. Following rest and recovery, the patient underwent another treadmill exercise trial at the identical work load used in the first trial, while receiving oxygen via the DODS adjusted to the equivalent setting. S_{pO_2} was recorded at the end of exercise.

If the S_{pO_2} with DODS was not equivalent to that with continuous flow, the patient underwent a third treadmill trial at the next higher DODS setting. If S_{pO_2} equivalency was still not achieved, the patient underwent a fourth exercise trial at the highest DODS setting.

The patients were encouraged to breathe normally for all trials and were not allowed to talk during the exercise trials. Differences in saturation and settings required to achieve those saturations were evaluated using paired *t* tests.

Results

At Rest

At rest, all 10 patients were adequately saturated via their continuous-flow prescription settings. Likewise, all the patients were adequately and equivalently saturated by the corresponding DODS settings (Figure 1). The group's mean ± SD saturations were 93.8 ± 1.9% with continuous flow and 93.7 ± 2.14% with DODS (that difference is nonsignificant; $p > 0.05$). Oxygen use with continuous flow was 1,700 ± 823 mL/min, whereas the DODS required 255 ± 123 mL/min to achieve the same S_{pO_2} ($p < 0.001$). Given an average at-rest respiratory rate of 15 breaths/min, the DODS oxygen-use efficiency advantage was 6.7-fold (ie, 1,700 vs 255 mL/min), which is both statistically significant and economically important.

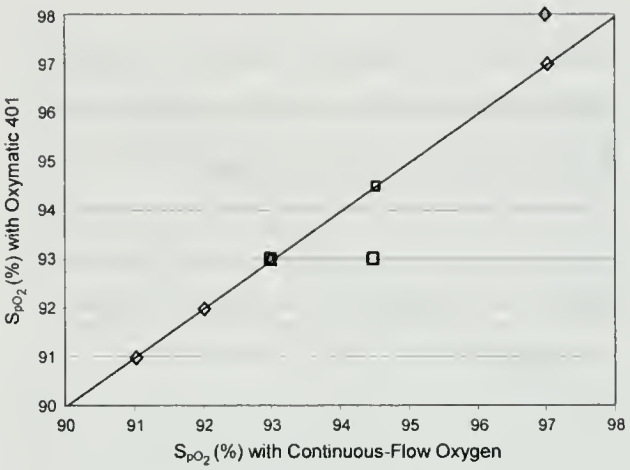

Fig. 1. At-rest oxygen saturation (measured via pulse oximetry [S_{pO_2}]) while using the Oxymatic 401 demand oxygen delivery system (DODS) versus while receiving continuous-flow oxygen. The DODS was equivalent to continuous flow for the same numbered settings.

During Exercise

All the patients tolerated the oxygen delivery equipment and the multiple exercise trials. Continuous-flow oxygen adequately saturated (ie, $S_{pO_2} > 90\%$) all patients except one, whose S_{pO_2} was 86% (Figure 2). Adjusted to the equivalent delivery setting, the DODS adequately saturated 6 patients, 4 of whom required the equivalent of continuous flow to achieve adequate saturation. The remaining 4 patients failed to achieve 90% S_{pO_2} at the corresponding settings. In the second exercise trial, with the DODS adjusted to the next higher setting, 3 of those patients achieved during-exercise $S_{pO_2} > 90\%$. The remaining patient (the one whose saturation was 86% on continuous-flow oxygen) matched his continuous-flow S_{pO_2}. Two patients required the highest DODS setting to achieve S_{pO_2} equivalent to their continuous-flow S_{pO_2}.

Figure 3 presents the same data as Figure 2 plus the data from the additional exercise trials at the higher DODS settings required to achieve the same S_{pO_2} as continuous flow. Single data points represent the patients whose DODS settings were equivalent to that with continuous flow. The lines represent the patients who required the higher DODS

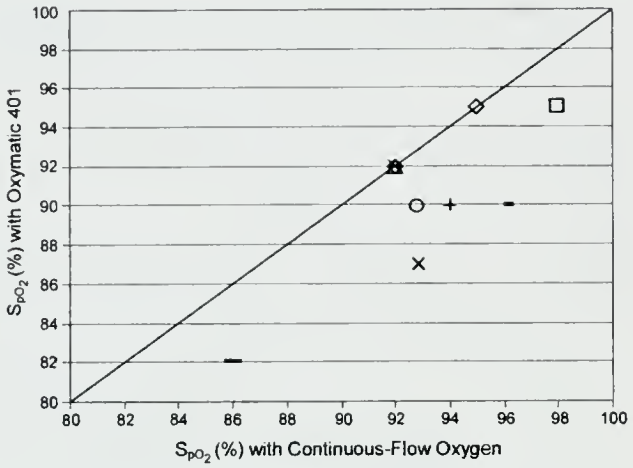

Fig. 2. Oxygen saturation (measured via pulse oximetry [S_{pO_2}]) during equivalent exercise work loads while using the Oxymatic 401 demand oxygen delivery system (DODS) versus while receiving continuous-flow oxygen. The DODS was equivalent to continuous flow for 4 patients. Six other patients did not achieve equivalent saturation.

settings. This figure illustrates the added benefit from the new settings (5 and 6).

At DODS settings corresponding to continuous flow, the mean S_{pO_2} of 90.5 ± 3.8% was significantly lower than the equivalent continuous-flow mean S_{pO_2} of 93.1 ± 3.1% ($p < 0.008$). At the higher settings the DODS achieved a mean saturation of 92.5 ± 2.8%, which was not significantly different ($p > 0.05$) from continuous flow. That is, the higher DODS settings achieved saturations equivalent to continuous flow. At corresponding settings, the DODS oxygen-use efficiency advantage was 5.6-fold, whereas at the higher setting the advantage was reduced to 4.3-fold. Hence, even when the DODS setting had to be increased, the oxygen savings was substantial and the medical benefits comparable to alternative delivery modalities.

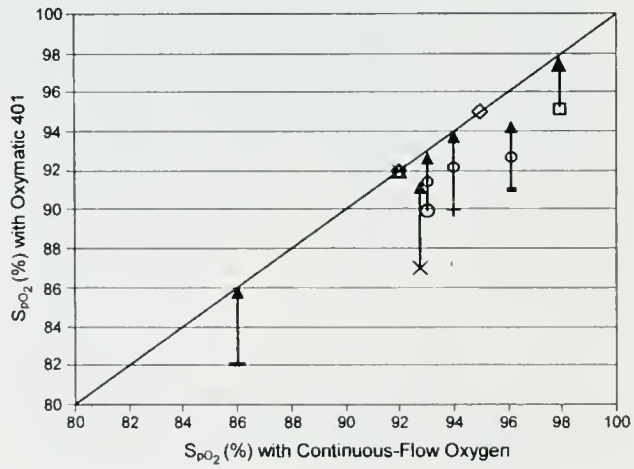

Fig. 3. As in Figure 2, oxygen saturation (measured via pulse oximetry [S_{pO_2}]) during equivalent exercise work loads while using the Oxymatic 401 demand oxygen delivery system (DODS) versus while receiving continuous-flow oxygen, but with the addition of higher DODS settings, as indicated by the arrows. The 6 patients who did not achieve equivalent saturation at the same DODS setting were equivalently saturated at the higher setting.

Discussion

This study reaffirms the efficacy of pulse DODS to deliver oxygen more efficiently while maintaining medically acceptable oxygen saturation. In at-rest patients the DODS achieved oxygen saturation equivalent to continuous flow at the equivalent setting. In exercising patients, saturation equivalency was not always achieved with the DODS at the same settings, but that was expected, because the patients were specifically chosen from a population who were known to experience exercise desaturation with DODS. Nevertheless, most patients were saturated > 90% at corresponding settings, and any comparative deficiency was corrected by raising the DODS setting to 5 or 6.

The reasons for the differences in at-rest and exercise saturation between DODS and continuous flow are both mechanical and physiologic. With DODS, the patient inhales a mixture of the 40–60 mL pure oxygen pulse and about 450 mL of atmospheric air (oxygen concentration 20.9%).[10] Those figures are the known mean tidal volume (V_T) for this patient group and the delivery pulse of the DODS. The DODS oxygen pulse is fixed in duration and volume, but the inspiratory breath pattern is highly variable. The oxygen pulse must arrive early in inspiration and must be fully included in the inspiratory flow to maximally benefit the patient. Continuous-flow oxygen also delivers a fixed flow, but the major difference is that the oxygen is always flowing and a large percentage of the oxygen is lost to the atmosphere and thus wasted. The efficiency of pulsed DODS relies on accurate timing to the patient's inspiration.

During exercise, respiratory rate and pattern change, depending on the pathophysiology of the chronic lung disease. In general, an increased respiratory rate is accompanied by faster inspiratory flow. A rapid inspiratory flow dilutes the oxygen pulse, whereas a slow inspiratory flow decreases dilution, which increases the fraction of inspired oxygen (F_{IO_2}) from the fixed-volume oxygen pulse.[11] However, a slower inspiratory flow could result in less air entrainment of the oxygen pulse and allow some of the oxygen to escape to the atmosphere. Therefore it is possible for the patient to breathe in a variety of patterns that could affect oxygenation in either direction. For example, pursed-lips breathing increases S_{pO_2} both on room air and on supplemental oxygen.[11] The complicated interactive effect of pursed-lips breathing on DODS delivery has several opposite influences on S_{pO_2}.

Exercise changes ventilation-perfusion matching and the ratio of dead space to V_T and thus impacts oxygenation. A pulse of oxygen being delivered under conditions of varying airflow pattern into a lung with changing ventilation-perfusion relationships would determine the percentage of oxygen participating in alveolar gas exchange.

Patients sometimes resort to mouth-breathing during exercise. Even though mouth-breathing is typically accompanied by some nasal air flow, a slower nasal flow tends to delay the onset of the DODS oxygen pulse by compromising the entrainment effect of the patient's inspiratory air flow. As a result some of the oxygen pulse can be lost to the atmosphere. In contrast, mouth-breathing during continuous-flow does not have that effect. In fact, mouth-breathers who have some nasal air flow often saturate well from continuous-flow oxygen.[11]

In a previous study we established the oxygenation benefit of early-inspiration pulse delivery.[12] We found that a 25 ms delay of pulse delivery measurably reduced S_{pO_2}. If some mouth-breathing occurring during exercise contributes to exercise desaturation, the patient could become dyspneic and resort to even more mouth-breathing. The result would create a "vicious cycle" of mouth-breathing, leading to desaturation leading to more mouth-breathing and an early termination to exercise. In one study, patients were allowed to severely desaturate during high-level exercise, probably creating such a self-reinforcing desaturation.[13] In the present study the most severely desaturating patients did resort to mouth-breathing, which may have contributed to exercise desaturation. However, this mouth-breathing was minimal, because no patient was subjected to maximal exercise. We encouraged patients to breathe normally by distracting them with one-way conversation, and we observed some mouth-breathing at the end of exercise.

One of the design modifications to the Oxymatic 401 was that the pulse volume was increased by 5 mL. Despite that larger pulse volume the at-rest oxygen savings was still nearly 7-fold over continuous-flow. During exercise several patients required a higher DODS delivery setting, which reduced the oxygen savings, but there was still a 4-fold advantage. The oxygen-use efficiency is important, because it bears on the portability of the oxygen system and impacts the economic burden of portable oxygen delivery. A lighter-weight DODS capable of maintaining oxygenation within medically prescribed limits will substantially impact quality of life for patients who desire to be more self sufficient.

The present standard of comparison for portable oxygen is the E-size cylinder and cart, which weighs about 22 lb and provides approximately 5.5 hours of oxygen at 2 L/min. The DODS, which holds 165 L of oxygen at 2,200 psi, weighs 4.5 lb and provides the at-rest patient approximately 9.2 hours of oxygen at Setting 2 (contrast 1.38 hours with 2 L/min continuous flow). DODS Setting 2 would meet the oxygenation needs of the majority of our patients at rest.

Several patients were adequately oxygenated on Setting 1, so their oxygen could last 18.4 hours at rest. If these patients continuously exercised at Setting 5, their oxygen would last 3.7 hours. Under usual conditions a patient would exert for 1 hour and be at rest for the remainder of the day. If that patient used DODS Setting 5 for that 1 hour of exercise, he or she would then have 6.2 hours remaining at Setting 2, or 12.4 hours remaining at Setting 1.

Any evaluation of the economics of home oxygen therapy must address both fixed and variable costs. The home medical equipment supplier operates within the boundaries of fixed reimbursement that is tied to the oxygen prescription and an escalating cost of providing the oxygen and the home service. Since the reimbursement is fixed, profitability can only be maintained by minimizing the cost of providing the service. One of the largest costs is the home visit. A stationary home oxygen concentrator requires a maintenance visit every 2–3 months. If the patient is using portable oxygen, the frequency of home medical equipment supplier visits can be as high as 3–4 times a month. At $30 to $50 per visit, the service cost alone could become greater than the reimbursement for that patient's portable oxygen.

An E-size cylinder provides about 5.5 hours of oxygen at 2 L/min. For a full day away from the stationary oxygen unit, the patient must therefore transport 2 E-size cylinders. A standard charge for refilling an E-size cylinder is $10. That calculates to $1.82 per hour of oxygen. For an 8-hour day the cost would be $14.56. If the patient were to use portable oxygen 10 days per month, the total costs for the E-size cylinder refills would be $145.60.

If a DODS were coupled to the E-size cylinder, with an oxygen savings of a modest 50%, the delivery time would be extended to 11 hours and the overall cost would decrease to $72.80 (under the foregoing assumptions), and fewer home deliveries would be necessary to meet the patient's needs. A higher oxygen savings of 70% would reduce the cost to about $40/month, which is about the level of Medicare reimbursement for the portable component of oxygen home therapy. Alternatively, the patient could be prescribed an aluminum D-size cylinder that holds 414 L of oxygen, weighs 10 lb, and provides 3.45 hours of oxygen at 2 L/min. A 50% savings would cause that cylinder to last for a full day, and a 70% savings would make that cylinder last 3–4 days and it would cost slightly more than the E-size cylinder at less than half the weight.

Presently, 4.5 lb mini-cylinders will last for a day and can be transfilled from an oxygen concentrator, freeing the oxygen dealer from regular home deliveries. Though the initial cost of such systems may run above $2,000 (a standard concentrator costs about $800), the savings in monthly

oxygen delivery to the home would rapidly compensate. Hence, combined technologies may lead to cost savings and a better quality of life for patients with chronic lung disease.

Not every patient will be a candidate for a pulsing oxygen-conserving device.[14] The variety of available stationary and portable oxygen systems gives patients and physicians choices, and knowledge of the available systems is recommended. That knowledge, along with understanding the patient's individual needs, will assist the clinician in making the best decision to enable the appropriate patients to maximize benefit from portable oxygen systems.

It must be emphasized that no patient should be prescribed oxygen without exercise-testing the patient with the oxygen device.[14] Portability is a highly desirable feature in oxygen systems for mobile patients, but portability should never be offered at the expense of failing to achieve adequate oxygen saturation. Fortunately, with the new higher-flow devices, that trade-off is much less of an issue.

Conclusions

The redesigned Oxymatic 401 DODS adequately oxygenates patients at rest and during exercise. In some cases the DODS setting must be raised 1 or 2 levels, which reduces the overall oxygen-use efficiency advantage but nevertheless provides the patient with a portable system with an extended service range that may accommodate the patient's active day on 1 cylinder. Because of the multifactorial impact of supplemental oxygen on the patient's physiology, psychological status, and quality of life, we recommended that every clinician be aware of the available portable oxygen systems and their reliability in maintaining saturation. All patients should be tested at rest and during usual exercise, using the oxygen system being prescribed, to determine appropriate delivery settings. Further studies are needed to better understand the dynamics of oxygen delivery under a variety of living conditions.

ACKNOWLEDGMENTS

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REFERENCES

1. Soffer M, Tashkin DP, Shapiro BJ, Littner M, Harvey E, Farr S. Conservation of oxygen supply using a reservoir nasal cannula in hypoxemic patients at rest and during exercise. Chest 1985;88(5):663–668.
2. Carter R, Williams JS, Berry J, Peavler M, Griner D, Tiep B. Evaluation of the pendant oxygen conserving nasal cannula during exercise. Chest 1986;89(6):806–810.
3. Heimlich HJ, Carr GC. Transtracheal catheter technique for pulmonary rehabilitation. Ann Otol Rhinol Laryngol 1985;94(5 Pt 1):502–504.
4. Christopher KL, Spofford BT, Brannin PK, Petty TL. Transtracheal oxygen therapy for refractory hypoxemia. JAMA 1986;256(4):494–497.
5. Tiep BL, Nicotra MB, Carter R, Phillips R, Otsap B. Low-concentration oxygen therapy via a demand oxygen delivery system. Chest 1985;87(5):636–638.
6. Tiep BL, Carter R, Nicotra B, Berry J, Phillips RE, Otsap B. Demand oxygen delivery during exercise. Chest 1987;91(1):15–20.
7. Carter R, Tashkin D, Djahed B, Hathaway E, Nictora MB, Tiep B. Demand oxygen delivery for patients with restrictive lung disease. Chest 1989;96(6):1307–1311.
8. Braun SR, Spratt G, Scott GC, Ellersieck M. Comparison of six oxygen delivery systems for COPD patients at rest and during exercise. Chest 1992;102(3):694–698.
9. Bower JS, Brook CJ, Zimmer K, Davis D. Performance of a demand oxygen saver system during rest, exercise, and sleep in hypoxemic patients. Chest 1988;94(1):77–80.
10. Tiep BL. Portable oxygen therapy with oxygen conserving methodologies. Monaldi Arch Chest Dis 1995;50(1):51–57.
11. Tiep BL. Electronic demand pulsed oxygen. In: Tiep BL, editor. Portable oxygen therapy: including oxygen conserving methodology. Mt Kisco NY: Futura; 1991.
12. Tiep BL, Christopher KL, Spofford BT, Goodman J, Worley PD, Macey SL. Pulsed nasal and transtracheal oxygen delivery. Chest 1990;97(2):364–368.
13. Roberts CM, Bell J, Wedzicha JA. Comparison of the efficacy of a demand oxygen delivery system with continuous low flow oxygen in subjects with stable COPD and severe oxygen desaturation on walking. Thorax 1996;51(8):831–834.
14. Standards for the diagnosis and care of patients with chronic obstructive pulmonary disease. American Thoracic Society. Am J Respir Crit Care Med 1995;152(5 Pt 2):S77–S120.

Impact of a Radio Frequency Management Information System on the Process and Timing of Providing Respiratory Care Services

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BACKGROUND: Although radio frequency (RF) systems have proliferated and are designed to simplify care delivery in many clinical settings, little information is available on the impact of such RF systems on the delivery of patient care. Having used a hand-held-device-based management information system in our Respiratory Therapy Section for 16 years, we assessed the impact of an RF system on the delivery of respiratory therapy (RT) services. METHODS: A single nursing unit dedicated to pulmonary and ear, nose, and throat care was selected for the RF system trial. Baseline (pre-RF) data were collected over 2 separate 1-month intervals (February 1999 and February 2000). The main outcome measures were (1) the amount of time needed at the beginning of the shift to organize and assign orders for RT services, (2) the time interval between notification of an RT consult order and completion of the RT consult, and (3) the time interval between notification of an RT treatment order and completion of the RT treatment. The activities required for organizing and assigning the orders were manually timed. Starting 6 weeks after therapists were trained to use the RF system, similar data were collected while using the RF system for two 1-month intervals (February and March 2001). RESULTS: The mean ± SD time interval between receiving an RT consult order and completing the consult was reduced from 7.8 ± 18.9 h to 2.8 ± 2.4 h ($p = 0.002$). The percentage of patients who waited longer than 8 hours between receipt of a consult order and completion of the consult decreased from 18% to 4.7% ($p = 0.026$). The total time required for organizing and assigning RT work was reduced from 81.6 min to 43.6 min. CONCLUSIONS: The RF system had several advantages over the hand-held-device-based system: (1) shorter interval between the order for and completion of an RT consult, (2) lower percentage of patients for whom the interval between the order and the consult exceeded 8 hours, and (3) less time required to make shift assignments. These results invite assessment of whether accelerated delivery of RT services confers clinical benefits. *Key words: radio frequency, management information system, respiratory care services.* [Respir Care 2002;47(8):893–897]

Introduction

Although radio frequency (RF) systems have proliferated with the intention to simplify and enhance delivery of care in many clinical settings, little attention has been given to evaluating the impact of such RF systems on the delivery of care. In the context of longstanding use (16 y) in our Section of Respiratory Therapy of a hand-held-device-based management information system, we wished to assess the impact of an RF system on the delivery of respiratory care services. To address this question and the lack of information in the available respiratory care literature, this observational study was undertaken to assess whether an RF management system would accelerate the delivery of respiratory care services to inpatients in the Cleveland Clinic Hospital.

Methods

Management Information System

Since 1986, orders for respiratory therapy (RT) services in the Cleveland Clinic Hospital have been processed using a management information system (CliniVision, Puritan-Bennett, St Louis, Missouri) that features a hand-held computer device carried by each respiratory therapist. The

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present study compares the timeliness of delivering RT services with that hand-held-device-based system and a system that features RF retrieval and delivery of new RT orders.

With the CliniVision hand-held-device-based system, the process of placing and delivering an order for an RT service includes 4 steps:

1. The physician writes an order in the medical chart for an RT service or consult.

2. The unit secretary transcribes the written order to a computerized order entry system (LastWord, IDX Systems, Burlington, Vermont) and places a copy of the printed order in the RT notebook on each hospital ward. If the therapist is present on the ward, he or she can retrieve the hard copy of the order and deliver the appropriate service.

3. The LastWord order entry system transfers the order to the CliniVision workstation in the Section of Respiratory Therapy office, where RT orders are organized and assigned to individual therapists. Each therapist comes to the office twice each shift (at the beginning and midway through) to upload assignments into the hand-held device and download the record of RT services delivered.

4. Upon returning to the hospital ward, each therapist delivers his or her assigned list of RT services.

The process of delivering RT orders with the RF system differs from the hand-held-device-based process in the following ways:

1. The therapist records, in a laptop computer (Amity, Mitsubishi, Japan) equipped with CliniVision software and an RF transceiver, each RT service delivered. Upon the therapist's command the laptop computer transmits via radio waves the record of therapies delivered to an "access point" base station installed on the ward, which communicates to the main workstation in the Respiratory Therapy Section office.

2. New orders for RT services during the shift are transmitted via radio waves to the therapist on the ward, rather than the therapist visiting the office to upload assignments from the main workstation.

Opportunities to accelerate the delivery of RT services using the RF system relate to the immediacy of RF transmission of new orders to therapists soon after their receipt from LastWord into the main CliniVision workstation. To receive new orders, the therapist must activate the appropriate function on the laptop computer. As a matter of policy with the RF system in this study, therapists were asked to look for new orders (ie, activate the function that seeks new orders) every 2 hours.

Outcome Measures

The main outcome measures assessed in this study were:

1. The amount of time needed at the beginning of the shift to organize and assign existing RT orders,

2. The time interval between placement of an RT consult order into the LastWord system and performance of the consult, and

3. The time interval between placement of an RT treatment order into the LastWord system and delivery of that treatment.

Hospital Ward Selection and Study Intervals

The study was conducted on a single hospital ward dedicated to caring for patients with ear, nose, and throat, and pulmonary illnesses at the Cleveland Clinic Hospital. This ward was selected because the volume of RT services was sufficiently large to occupy a single therapist for the entire shift, thereby avoiding the need to cover multiple hospital wards using multiple hand-held devices or RF computers. Also, the large volume of respiratory therapies administered on this ward allowed collection of data from a large number of patients while requiring installation of RF system hardware (access point base station, antennae, etc) on only a single ward.

Baseline data from the hand-held-device-based system were collected during February 1999 and February 2000. These baseline data were selected for the same calendar months as the RF system study period in order to minimize effects from seasonal variation in the volume and types of respiratory care services delivered.

Collection of data with the RF system began after a 6-week training period in which the therapists on the ear, nose, and throat/pulmonary ward received instruction and had practice with the RF system. February and March 2001 were the data collection periods for the RF system.

Shift Assignment Times

For the hand-held-device-based (CliniVision) process, the mean times required for each activity in making shift assignments (eg, uploading to and downloading from hand-held devices and calculating and assigning work loads) were determined during 3 separate assignment periods. The comparable times for the RF system were determined by subtracting the time intervals using the RF system from the intervals using the hand-held-device-based system. For example, the average time required to calculate and assign work loads using the hand-held-device-based system was 31.6 min. Two minutes of that time were used to page therapists and obtain updated treatment numbers. Therefore, because the RF system does not require paging therapists, 2 minutes were deducted from the measured hand-held-device-based system time, which made the RF-system time for assigning work loads 29.6 minutes (Table 1).

Table 1. Time Required per Shift

Activity	HH	RF	Difference*
Calculate and assign work loads	31.6	29.6	−2.0
Upload data to hand-held device	28.3	0	−28.3
Download data from hand-held device	10.0	0	−10.0
Print reports	11.7	14.0	2.3
Total for shift assignment	81.6	43.6	−38.0

HH = hand-held-device-based system
RF = radio frequency management information system
*Difference between HH and RF systems. Negative number indicates less time needed with the RF system
Values are mean minutes.

Statistical Analysis

Statistical analyses were conducted with commercially available software (SigmaStat version 2.03, SPSS Science, Chicago, Illinois). Nonparametric tests (Mann-Whitney and chi-square) were used because of the non-normal distribution of the data. Values of $p < 0.05$ were deemed statistically significant.

Results

Table 2 presents the data from the 115 patients during February 1999 and February 2000 (ie, the CliniVision system period) and from the 96 patients during February and March 2001 (ie, the RF system period). Comparison of baseline features showed that the RF group was slightly younger (median age 57 vs 62 y, $p = 0.030$) but that there were no significant differences in the distribution of sex or underlying disease.

As shown in Table 3, the RF system was associated with accelerated delivery of RT services. Specifically, the mean interval between receipt of a consult order and performance of the consult decreased by 5 hours (from 7.8 to 2.8 h, $p = 0.002$) and the percentage of patients who waited > 8 hours for completion of an RT consult decreased from 18.0% to 4.7% ($p = 0.026$).

Nonsignificant trends suggesting accelerated delivery of respiratory treatments included a shortened interval (by mean 1 h) between notification of RT orders and initiation of therapy (5.9 vs 4.9 h, $p = 0.160$) and a slightly smaller percentage of patients who waited > 8 hours to receive respiratory treatments (17.4% vs 14.6, $p = 0.716$). The RF system was also associated with less time required to complete a shift assignment (mean 81.6 vs 43.6 min; see Table 1). Finally, because the RF system obviates therapist visits to the office to upload newly received orders, the time for therapist travel and for printed report generation in the office was eliminated.

Discussion

Our main finding is that the RF management information system for RT orders was associated with accelerated performance of RT consults and faster completion of RT shift assignments. Other trends suggesting accelerated delivery of RT services were also evident (eg, a mean 1-hour shortening of the interval between receipt of orders and treatment delivery).

Despite considerable commercial enthusiasm to implement RF systems in health care, review of the literature indexed in MEDLINE (1966 through September 2001), the Cumulative Index to Nursing and Allied Health Literature (CINAHL), and the Health STAR database, using combinations of the keywords "radio," "radiofrequency," "delivery," "services," and "health care," showed that the impact of RF systems on the delivery of care has received little formal attention. Specifically, only 7 citations were found using that search strategy.[1–7] Chin[1] summarized experience with RF systems in nursing. He cited the experience from Good Samaritan Hospital (Dayton, Ohio), in which implementing a wireless point-of-care system reportedly saved nurses 30–45 min per shift by eliminating inefficiencies in recording drug administration.

In another review of experience with RF systems in health care, Grimm[2] reported an experience from St Clair Memorial Hospital (Pittsburgh, Pennsylvania) in which a wireless point-of-care documentation system for nurses was associated with a 33% decrease in nurses' time to document and a concomitant $600,000 annual savings.

In a report regarding the use of RF systems for delivering physical therapy services at Good Shepherd Medical Center (Longview, Texas), Levine[3] reported that the RF system was associated with fewer therapist transcription errors and improved access to key clinical information.

Hammer et al[4] reported experience with a bedside RF-equipped computer system in performing psychiatry consultations. Documentation time was reportedly halved, compared to the prior practice of hand-writing notes.

Regarding RF systems in respiratory care, Chin[1] cited widespread implementation at Ohio State University Medical Center and, to our knowledge, reported the only available data regarding the impact of RF on RT care delivery. Specifically, at St Luke's Episcopal Hospital (Houston, Texas), transition from a desktop-computer-based to a wireless, laptop-based management information system was associated with a 10% increase in therapist productivity, because therapists were able to see more patients daily. Our findings are consistent with the trend in these earlier reports, of accelerated care delivery with RF systems. At the same time, the anecdotal nature of available observations and the paucity of studies underscore the need for additional studies.

Table 2. Characteristics of the Study Population

	Age* (y)			Sex		
	Mean	SD	Median	Female (%)	Male (%)	*n*
Pre-RF Group	61.9	16.6	65	54 (47)	61 (53)	115
RF Group	57.1	17.6	55	53 (55)	43 (45)	96

Diagnosis: Pre-RF Group (n = 115)

	Pneumonia	COPD	Asthma	Lung Cancer	Other	Total	%
Pulmonary	19 (36%)	12 (23%)	5 (9%)	6 (11%)	11 (21%)	53	46
ENT	—	—	—	—	—	27	24
Other	—	—	—	—	—	35	30

Diagnosis: RF Group (n = 96)

	Pneumonia	COPD	Asthma	Lung Cancer	Other	Total	%
Pulmonary	13 (30%)	13 (30%)	8 (18%)	1 (2%)	9 (20%)	44	46
ENT	—	—	—	—	—	20	21
Other	—	—	—	—	—	32	33

RF = radio frequency management information system
COPD = chronic obstructive pulmonary disease
ENT = ear, nose, and throat
*There was a statistically significant difference in median values between the 2 groups (p = 0.030 by Mann-Whitney test): the RF group is slightly younger. There were no significant differences in gender or diagnostic groups.

Table 3. Time Intervals from Receipt of Respiratory Therapy Orders to Delivery of RT Services

	HH	RF	p	Test
Number of consult evaluations	82	64	NA	NA
Mean time (h ± SD) from notification to consult evaluation	7.8 ± 18.9	2.8 ± 2.4	0.002	Mann-Whitney
Number and percent of patients who waited > 8 h for evaluation	15 (18.0%)	3 (4.7%)	0.026	chi-square
Number of patients with respiratory therapy orders	115	96	NA	NA
Mean time (h ± SD) from notification to start of therapy	5.9 ± 8.4	4.9 ± 7.8	0.160	Mann-Whitney
Number and percent of patients who waited > 8 h for therapy	20 (17.4%)	14 (14.6%)	0.716	chi-square

HH = hand-held-device-based system
RF = radio frequency management information system
NA = not applicable

Though our results suggest accelerated service delivery with an RF system, several shortcomings of the present study are noteworthy. First, because our analysis did not assess clinical outcomes that might be affected by accelerated (or delayed) delivery of RT treatments, we are unable to ascribe clinical benefits to the accelerated delivery observed here. We speculate that quicker receipt of RT treatments enhances patient comfort and satisfaction and may confer other clinical benefits, but the issue requires further study.

Second, although we observed accelerated delivery with the RF system, it is important to point out that other factors could have affected the time interval between placement of the order and receipt of the treatment. For example, using our hospital's computerized order entry system as described, this interval would be affected by the time elapsed between the physician's writing the order and the unit secretary's entering the order into the computer. Although audit experience in our hospital suggests that this interval is short, the specific intervals were not assessed during the study periods considered here.

Other practices that could affect the intervals between order, receipt, and delivery of respiratory care services

include the frequency with which therapists visited the Respiratory Therapy Section office (when using the hand-held devices). According to our practice in the current study, therapists brought their hand-held devices to the office for uploading at mid-shift, approximately 4 hours after receiving the first set of RT orders. Clearly, a policy under which therapists visit the office more frequently during the shift could have opposing effects on the speed of delivering RT services. On the one hand, new orders might be uploaded to the hand-held devices sooner during the shift. On the other hand, more frequent visits to the office during the shift could slow the delivery of services, as therapists spend more time in transit, away from the bedside. Given the observational nature of our study and our established practice of one mid-shift visit to upload new orders to the hand-held device, this study did not attempt to assess the "ideal" visit frequency to minimize the interval between order placement and treatment delivery.

Also, the interval between order placement and delivery with the RF system would be influenced by the frequency with which therapists request new orders via their laptop computers. In the current study, this request frequency was every 2 hours, but it seems possible that the delivery of orders could be accelerated further by therapists' requesting orders more frequently.

Conclusions

Overall, our results show that, compared with a hand-held-device-based system, use of an RF system for processing and delivering RT orders was associated with several benefits:

1. A shorter interval between the order for and completion of an RT consult,
2. A lower proportion of patients for whom the interval between the order and the consult exceeded 8 hours, and
3. Less time required for supervisors to make shift assignments.

In the context that little attention has been given to the impact on care delivery of this appealing but expensive new technology, our results certainly invite further critical assessment of the clinical benefits associated with RF management information systems and the effects of accelerated delivery of RT services.

REFERENCES

1. Chin TL. Is wireless technology ready to roll? Health Data Manag 1998;6(5):78–82, 84–86, 88–89.
2. Grimm B. Healthcare unplugged. Hospitals embrace mobile computing and wireless communications. Healthc Inform 1998;15(3): 81–82.
3. Levine R. Avoid the paper chase: real-time, wireless patient documentation helps therapists save both time and money. Healthc Inform 1999;16(1):73–74.
4. Hammer JS, Strain JJ, Friedberg A, Fulop G. Operationalizing a bedside pen entry notebook clinical database system in consultation-liaison psychiatry. Gen Hosp Psychiatry 1995;17(3):165–172.
5. Hisle M. Universal broadband communications creates new options for healthcare networks. J Healthc Inform Manag 2000;14(2):71–84.
6. Kohli J, Brody G, Melvin C, Muhlstein L. Wireless communications in the delivery of healthcare care. In: Proceedings of the 1995 Annual HIMSS Conference, Feb 12–16,1995. Volume 4. Chicago: HIMSS 1995:221–234.
7. Hanley J. Telemetry in health care. Biomed Eng 1976;11(8):269–272.

The Effects of Tidal Volume Demand on Work of Breathing During Simulated Lung-Protective Ventilation

Richard H Kallet MSc RRT, James A Alonso RRT, Martha Diaz RRT, Andre R Campbell MD, Robert C Mackersie MD, and Jeffrey A Katz MD

BACKGROUND: Lung-protective ventilation (LPV) can result in a ventilator tidal volume (V_T) below patient V_T demand, which may elevate work of breathing (WOB). Increasing the ventilator inspiratory flow may not sufficiently reduce WOB, because the patient's flow-time requirements may exceed the ventilator's flow-time delivery pattern. We investigated (1) the effects of V_T demand on WOB during LPV and (2) which ventilator pattern best reduced WOB while achieving LPV goals. METHODS: A standard WOB lung model simulated assisted breathing. Using 3 ventilators (Hamilton Veolar, Hamilton Galileo, and Dräger Evita 2 dura), we tested volume-control ventilation with a constant flow pattern (VCV-CF), volume-control ventilation with a decelerating flow (VCV-DF), and pressure-control ventilation (PCV). Simulated V_T demand was increased from 50–125% of the ventilator-delivered V_T (400 mL) as ventilator inspiratory time (T_I) was decreased (0.95, 0.80, 0.65, and 0.45 s) relative to simulated T_I (0.8 s). WOB was measured with a pulmonary mechanics monitor. RESULTS: During VCV-CF and VCV-DF, a V_T demand of ≥ 100% drastically increased WOB, attributable to imposed WOB from the inspiratory valve. Increasing inspiratory flow by using the decelerating flow pattern and/or decreasing T_I reduced WOB, but generally not to normal levels. "Double-triggered" breaths, with excessive V_T delivery, often occurred when ventilator T_I was well below simulated T_I. PCV was most effective in reducing WOB, but V_T delivery exceeded the LPV target unless T_I was reduced. CONCLUSIONS: Given our dual goals of reducing both WOB and V_T during LPV, VCV-DF with relatively brief T_I appeared to be the best option, followed by PCV with a relatively brief T_I. *Key words: acute lung injury, assisted mechanical ventilation, imposed work of breathing, inspiratory flow rate, inspiratory flow waveform, lung model, lung-protective ventilation, tidal volume, work of breathing.* [Respir Care 2002;47(8):898–909]

Introduction

During assisted mechanical ventilation (AMV) flow asynchrony increases the work of breathing (WOB) performed by the patient[1–4] because (it is thought) the ventilator fails to push the inspired gas at the same flow as the inspiratory muscles attempt to pull gas into the lungs.[1,4] In this "push-pull system," total WOB is thought to remain relatively constant, with only the distribution of work between the patient and the ventilator varying because of patient effort.[1] Yet it is also speculated that an "additional inspiratory load" occurs when patient flow demand exceeds the ventilator flow delivery and "the patient tries to accelerate gas against the inspiratory resistance of the ventilator circuit,"[1] which implies that the ventilator creates imposed WOB (WOB_{imp}) based on patient effort. However, this theory has not been confirmed by actually measuring WOB_{imp} during AMV.

Increased WOB from flow asynchrony occurs because patient flow demand approximates the contractile velocity of the inspiratory muscles.[5] By extension, tidal volume

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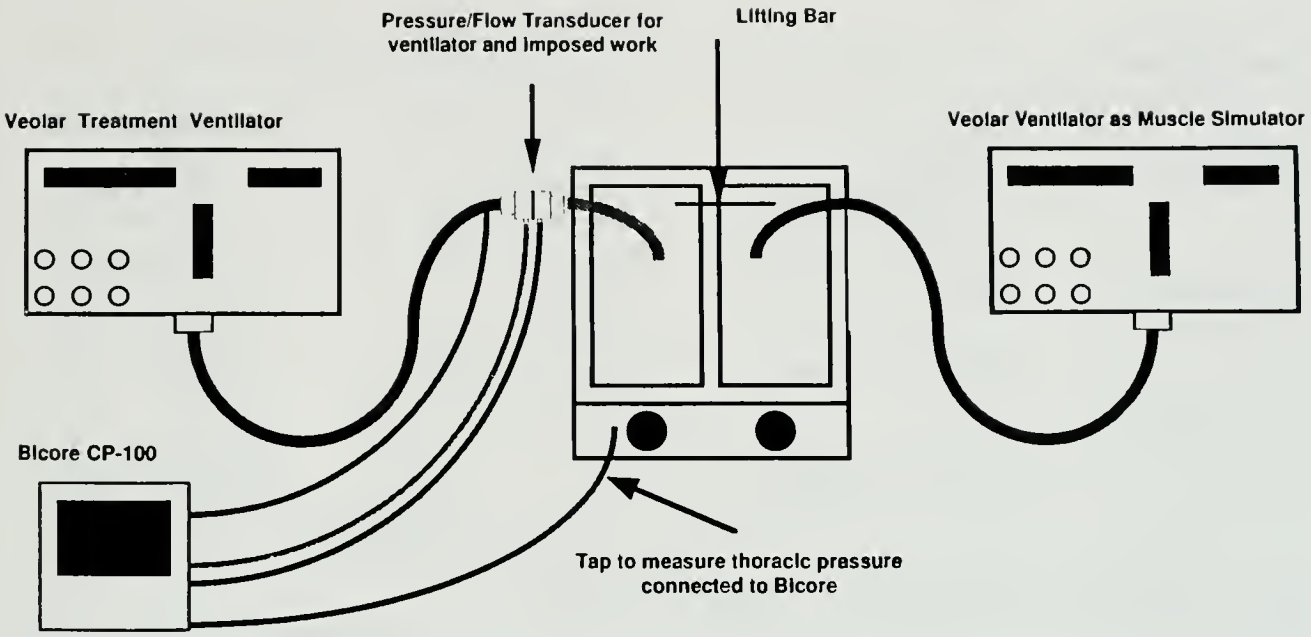
Pressure/Flow Transducer for ventilator and imposed work
Lifting Bar
Veolar Treatment Ventilator
Veolar Ventilator as Muscle Simulator
Bicore CP-100
Tap to measure thoracic pressure connected to Bicore

Fig. 1. A standard work of breathing lung model with a dual-compartment test lung was used to represent the "push-pull" concept of assisted mechanical ventilation. The right-side compartment simulated the inspiratory muscles and was linked by a lifting bar to the left-side compartment, which represented the thorax. Displacement of the thoracic compartment by the inspiratory muscle compartment (representing the "pull") simulated spontaneous breathing effort. The treatment ventilator, which represented the "push", was attached to the thoracic compartment and could displace it independently of the muscle compartment. Bicore CP-100 = pulmonary mechanics monitor. (From Reference 12, with permission.)

(V_T) demand reflects global inspiratory muscle shortening.[5] Hence, WOB_{imp} may occur during AMV when ventilator V_T delivery does not meet patient V_T demand. Patient-ventilator V_T mismatching may become a problem when lung-protective ventilation (LPV) is used to treat acute respiratory distress syndrome (ARDS).[6] LPV requires a V_T between 4 and 7 mL/kg,[7–10] which can be less than the spontaneous V_T reported in ARDS patients (300–400 mL).[1,11] Recently, a case of apparent V_T mismatching resulting in acute alveolar edema was reported in a patient enrolled into the National Institutes of Health ARDS Network study.[12]

Does a specific manipulation of the ventilator flow waveform and/or inspiratory time (T_I) effectively reduce WOB during V_T mismatching? Both pressure-control ventilation (PCV)[11,13] and volume-assured pressure support ventilation[4,14] are associated with less patient WOB than the constant flow pattern commonly used during volume-control ventilation (VCV). But the difference in efficacy between VCV with a fixed decelerating flow pattern and the variable decelerating flow pattern of PCV and volume-assured pressure support has not been studied. Alternatively, increasing the inspiratory flow ($\dot{V}_I$) during VCV with constant flow pattern (VCV-CF) by shortening ventilator T_I causes similar reductions in patient effort compared to pressure-limited ventilation.[15] Yet if the patient's T_I exceeds that of the ventilator, "double-triggering" (ie, 2 ventilator breaths for a single breathing effort) may occur[16] and frustrate attempts to achieve LPV.

Methods

We used a standard WOB lung model[17] to imitate patient-ventilator interactions during AMV and thereby measured the effects of simulated patient V_T demand on simulated patient WOB (WOB_{sim}) during LPV. We investigated 3 ventilator flow waveforms (VCV-CF, VCV with decelerating flow pattern [VCV-DF], and PCV) and 4 ventilator T_I settings. We sought to find which combination of ventilator flow waveform and T_I could best minimize WOB_{sim} while maintaining a reasonable degree of LPV.

Apparatus

A dual-compartment Model 1600 Training Test Lung (Michigan Instruments, Grand Rapids, Michigan) was used (Fig. 1). One compartment simulated the inspiratory muscles and was connected to a ventilator (Veolar, Hamilton, Reno, Nevada). The other compartment simulated a patient's thorax. The compartments were linked by a lifting bar so that the positive pressure developed during inflation of the inspiratory muscle compartment lifted the thoracic compartment and created a corresponding negative pressure. The rate increase in negative pressure ($\Delta P/\Delta T$) cre-

Table 1. Protocol 1

Treatment Ventilator Settings	VCV-CF	VCV-DF	PCV
T_I = 0.95 s f = 20 breaths/min V_T = 400 mL (CMV)	Muscle Simulator Settings T_I = 0.80 s f = 24 breaths/min V_T: 200, 300, 400, 500 mL	Muscle Simulator Settings T_I = 0.80 s f = 24 breaths/min V_T: 200, 300, 400, 500 mL	Muscle Simulator Settings T_I = 0.80 s f = 24 breaths/min V_T: 200, 300, 400, 500 mL
T_I = 0.80 s f = 20 breaths/min V_T = 400 mL (CMV)	Muscle simulator settings constant	Muscle simulator settings constant	Muscle simulator settings constant
T_I = 0.65 s f = 20 breaths/min V_T = 400 mL (CMV)	Muscle simulator settings constant	Muscle simulator settings constant	Muscle simulator settings constant
T_I = 0.45 s f = 20 breaths/min V_T = 400 mL (CMV)	Muscle simulator settings constant	—	Muscle simulator settings constant

VCV-CF = volume-control ventilation with constant flow pattern
VCV-DF = volume-control ventilation with decelerating flow pattern
PCV = pressure control ventilation
T_I = inspiratory time
f = respiratory rate
V_T = tidal volume
CMV = controlled mechanical ventilation

ated in the thoracic compartment represented transmitted muscle pressure to the alveolar surface. The resulting $\dot{V}_I$ and V_T measured during displacement of the thoracic compartment served as an analog for patient demand. The thoracic compartment was attached to a treatment ventilator (either a Veolar [Hamilton Medical, Reno, Nevada], Galileo [Hamilton Medical, Reno, Nevada], or an Evita 2 dura [Dräger, Telford, Pennsylvania]). In order to model the "push-pull system" of patient-ventilator interactions during AMV, the thoracic compartment could be displaced independently of the inspiratory muscle compartment. Thus, when the treatment ventilator "pushed-up" the thoracic compartment faster than the inspiratory muscle simulator could "pull-up" the thoracic compartment, the WOB performed by the simulator on the thoracic compartment was reduced. In this paper "simulated patient demand" (for flow or V_T) is referred to as "demand." The terms "ventilator" and "ventilator delivery" refer to the treatment ventilator and its delivery of flow or V_T. The "inspiratory muscle simulator" is the ventilator that powered the inspiratory muscle compartment.

The compliance of both test lung compartments was set at 27 mL/cm H_2O, and the resistance of each unit was equal and created by using two (8.0 mm internal diameter) endotracheal tubes with (15 mm) adapters at both ends. The airways resistance, measured at a constant $\dot{V}_I$ of 60 L/min, was 5.0 cm H_2O/L/s. The values for compliance[6,10,18] and resistance[18] were within the range described for patients with ARDS.

Measurements and Calculations

All variables were measured with a previously validated[19,20] pulmonary mechanics monitor (Bicore CP-100, VIASYS Healthcare/Bear Medical Systems, Palm Springs, California). The monitor and transducers were calibrated prior to each experiment. Airway pressure (P_{aw}) and $\dot{V}_I$ were measured at the Y-adapter with a flow transducer (VarFlex, VIASYS Healthcare/Bear Medical Systems, Palm Springs, California). V_T was calculated from the integrated measurements of $\dot{V}_I$ and T_I and excluded circuit compression volume. T_I was calculated as:

$$60 \text{ s/set f} \times T_I \text{ fraction}$$

in which f is the respiratory rate (in breaths/min) and T_I fraction is the ratio of inspiratory time to total breathing cycle time. T_I was confirmed by calibrated scalar flow tracings.

WOB_{sim} was the work performed by the inspiratory muscle simulator to displace the thoracic compartment. It provided a gross representation of patient WOB to overcome the elastic and resistive properties of the thorax, in addition to any WOB_{imp} from the ventilator inspiratory valve. WOB_{sim} required measuring the thoracic compartment pressure by attaching a neonatal extension tube (SmartCath, VIASYS Healthcare/Bear Medical Systems, Palm Springs, California) to the pressure-tap of the compartment (see Fig. 1). WOB_{sim} was calculated by the pulmonary mechanics monitor as the area within the thoracic

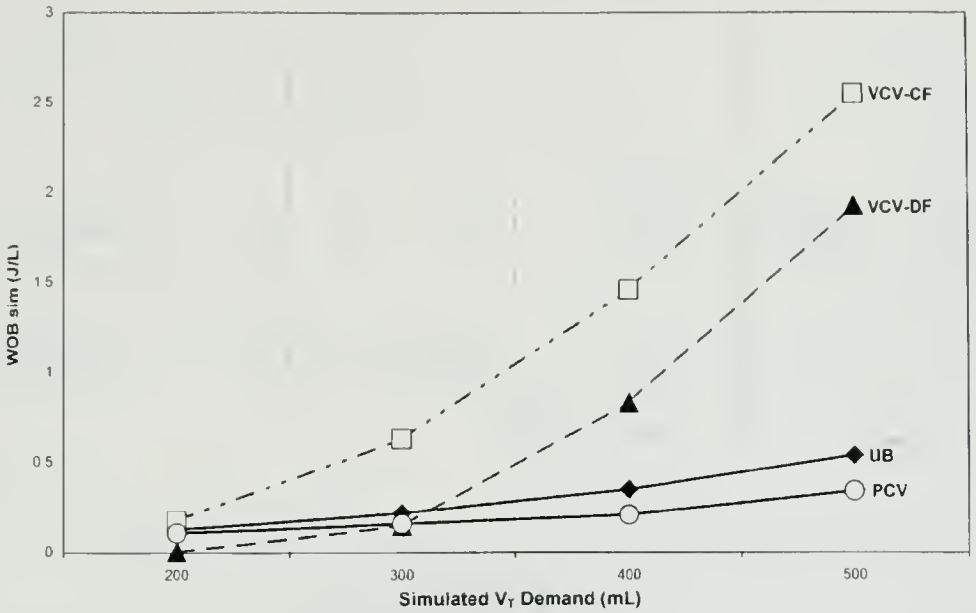

Fig. 2. Simulated work of breathing (WOB_{sim}) during assisted mechanical ventilation (inspiratory time 0.95 s) and simulated unassisted breathing (UB) as a function of tidal volume (V_T) demand (Protocol 1). WOB_{sim} rose substantially at 100% V_T demand during volume-control ventilation with constant flow pattern (VCV-CF) and during volume-control ventilation with decelerating flow pattern (VCV-DF). During pressure-control ventilation (PCV) WOB_{sim} paralleled the rise that occurred with UB, but PCV consistently reduced WOB_{sim} to below the UB level. These general trends in WOB_{sim} were also observed as ventilator inspiratory time was decreased to 0.80 s and 0.65 s.

compartment pressure-V_T curve when thoracic compartment pressure was below the end-expiratory baseline.[19]

WOB performed by the ventilator (WOB_{vent}) to displace the thoracic compartment was measured by the flow transducer placed at the Y-adapter of the ventilator circuit. WOB_{vent} was calculated by the pulmonary mechanics monitor as the area within the P_{aw}-V_T curve when the P_{aw} was above the end-expiratory baseline.[19] WOB_{imp} was the work performed by the inspiratory muscle simulator to overcome circuit resistance due to the behavior of the ventilator's inspiratory valve, based on clinician settings of T_I, V_T, flow waveform, or P_{aw}. WOB_{imp} required measuring P_{aw} with the neonatal extension tube located at the Y-adapter of the ventilator circuit. WOB_{imp} was calculated as the area within the P_{aw}-V_T curve when the P_{aw} remained below the end-expiratory pressure level.[17] WOB was expressed in joules per liter of ventilation (J/L).

Protocols

Protocol 1: The Effects of Varying V_T Demand and Peak $\dot{V}_I$ Delivery on WOB_{sim}. This protocol was designed to study 4 questions:

1. What are the effects of increasing V_T demand on WOB_{sim} with different ventilator flow waveforms?

2. How effective is incrementally increasing peak $\dot{V}_I$ delivery with VCV in reducing WOB_{sim}? Ventilator peak $\dot{V}_I$ was increased by reducing T_I in steps (0.95, 0.80, 0.65 and 0.45 s) (Table 1).

3. Do the same stepwise T_I reductions during PCV reduce WOB_{sim} while limiting the potential "overshoot" in V_T delivery?

4. Is WOB_{imp} created during conditions of peak $\dot{V}_I$ and V_T mismatching?

The ventilator was set to deliver a 400 mL V_T to the thoracic compartment at a mandatory respiratory rate of 20 breaths/min. This approximated a V_T of 6 mL/kg predicted body weight, reported in 2 LPV studies.[6,10] During PCV, the target V_T of 400 mL was achieved by setting the pressure control level to the end-inspiratory plateau pressure achieved during VCV. A pressure-rise time of zero was used during PCV. With the Veolar and Galileo ventilators, trigger sensitivity was set at –1 cm H_2O. With the Evita 2 dura ventilator a nonbiased flow trigger level of 5 L/min was set. For the Veolar and Galileo ventilators a full exponential decelerating flow waveform was used for VCV-DF. No positive end-expiratory pressure was set on any ventilator, and expiration was passive and completed prior to the next triggered breath so that no intrinsic positive end-expiratory pressure was present during any experiment. No humidification device was used in any circuit.

The inspiratory muscle simulator generated 4 levels of V_T demand (200, 300, 400, and 500 mL) in the thoracic compartment. This represented 50, 75, 100, and 125% of the ventilator-delivered V_T under controlled mechanical ventilation (CMV) conditions (ie, time-triggered breaths by the treatment ventilator while the inspiratory muscle

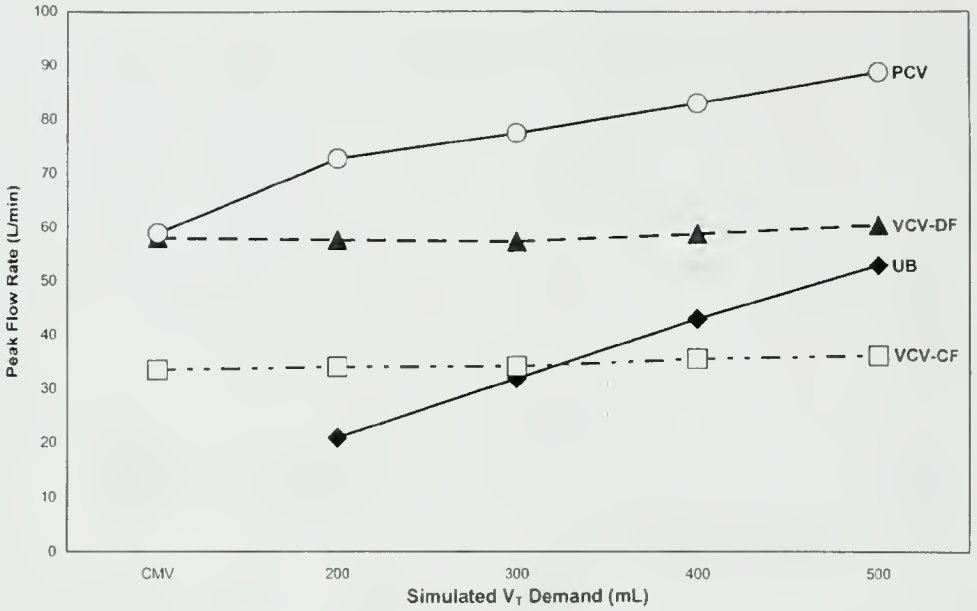

Fig. 3. Peak inspiratory flow ($\dot{V}_I$) during assisted mechanical ventilation (inspiratory time 0.95 s) and simulated unassisted breathing (UB) as a function of tidal volume (V_T) demand (Protocol 1). Compared to Figure 2, the increase in simulated work of breathing as V_T demand increased during volume-control ventilation with constant flow pattern (VCV-CF) (300 mL) and volume-control ventilation with decelerating flow pattern (VCV-DF) (400 mL) occurred despite ventilator peak $\dot{V}_I$ either matching or exceeding flow demand. During pressure-control ventilation (PCV), peak $\dot{V}_I$ rose in parallel with the flow demand of UB.

simulator was inactive). The inspiratory muscle simulator was set in the VCV mode at a respiratory rate of 24 breaths/min, with a T_I of 0.80 s. A modified sine wave was used so that peak $\dot{V}_I$ occurred at approximately 0.2 s (25% of T_I). This resulted in 4 levels of peak flow demand: 21, 32, 43, and 53 L/min.

Tests with CMV were used to establish baseline WOB_{vent} and to verify ventilator $\dot{V}_I$ and V_T delivery with each flow pattern, mode, and T_I setting. Likewise, baseline measurements of WOB_{sim}, flow, and V_T demand were verified during simulated unassisted breathing (ie, with the ventilator disconnected from the thoracic compartment of the model). In addition, WOB_{imp} was measured during unassisted breathing in the continuous positive airway pressure mode with an end-expiratory pressure of 0 cm H_2O. Afterwards, AMV experiments were done for each flow waveform and ventilator-set T_I. VCV-DF could not be used at a T_I of 0.45 s, because the ventilator defaulted to a constant flow pattern to maintain V_T delivery. In addition, the 400 and 500 mL V_T demand portions of the protocol were repeated with different ventilators (Galileo and Evita 2 dura). Because the Evita 2 dura ventilator does not provide a VCV-DF option, WOB was measured only during VCV-CF and PCV.

Protocol 2: Constant Peak $\dot{V}_I$ Demand at Different Levels of V_T Demand. Increasing V_T demand at a fixed T_I also raises peak flow demand. Therefore, increasing flow demand (rather than V_T demand) may explain differences in WOB_{sim} and WOB_{imp}. To assess this potentially confounding variable we examined whether the effects of increasing V_T demand on WOB_{sim} could be isolated from that of increasing flow demand. To do this without introducing intrinsic positive end-expiratory pressure into the model, a traditional-size V_T of 800 mL was set on the ventilator at a respiratory rate of 12 breaths/min and T_I = 1.0 s. The inspiratory muscle simulator was adjusted to create the same peak flow demand (72 L/min) at 2 levels of V_T demand: 800 and 500 mL (100% and 60% V_T demand, respectively). The 500 mL V_T demand test required changing the muscle simulator respiratory rate from 15 to 24 breaths/min and simulator T_I from 1.0 to 0.58 s.

Data Analysis

A sample size of 12 breaths was used for each variable. Multiple comparisons were made using Tukey-Kramer tests after either repeated measures or one-way analysis of variance.[21] Paired comparisons were made using the *t* test. Statistical analysis was done with commercially available software (GraphPad InStat, version 3.0, GraphPad Software, San Diego, California). Differences were considered significant if $p < 0.05$.

Results

For each variable tested in each protocol, there was a very narrow dispersion of data, and the average coefficient of variation was < 0.05. Therefore, for simplicity, all data

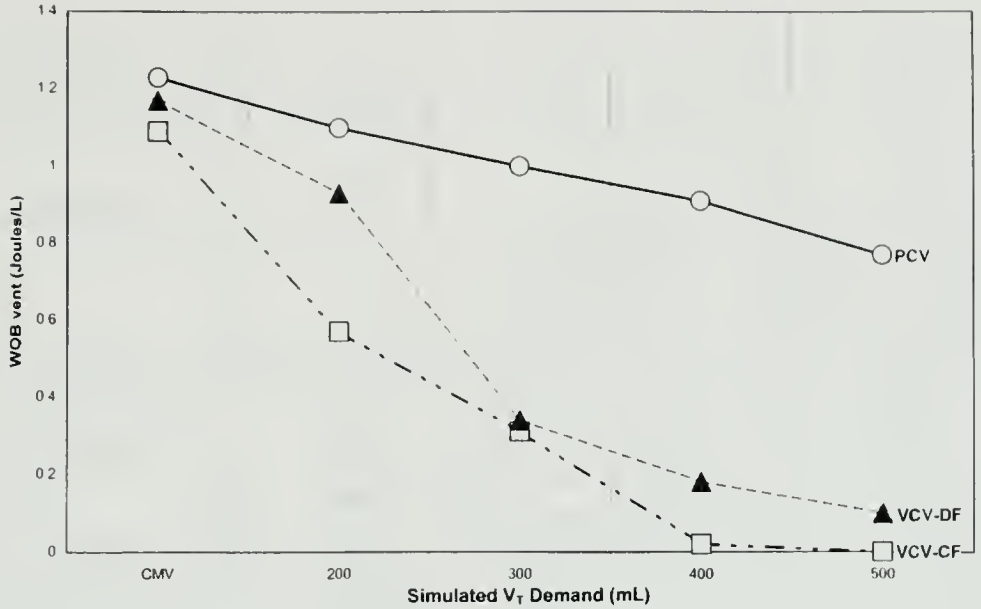

Fig. 4. Work of breathing performed by the ventilator (WOB_{vent}) during assisted mechanical ventilation (inspiratory time 0.95 s) as a function of tidal volume (V_T) demand (Protocol 1). As V_T demand increased there was a precipitous decline in WOB_{vent} during volume-control ventilation with constant flow pattern (VCV-CF) and volume-control ventilation with decelerating pattern (VCV-DF), which corresponded with the rise in simulated work of breathing (WOB_{sim}). In contrast, during pressure-control ventilation (PCV), WOB_{vent} declined more gradually and reflected the more gradual rise in WOB_{sim}.

are reported as mean values. More importantly, a majority of the differences were statistically significant, though some differences were clinically unimportant. To determine what constituted a clinically important difference in WOB, we reviewed several studies[1,11,13,14,22–25] that compared patient WOB in which either the pre-set peak $\dot{V}_I$ or flow pattern was manipulated. Based on those studies we calculated a treatment effect[26] of 0.15 J/L and used that value as a threshold for determining the clinical relevance of our results.

Protocol 1: The Effects of Varying V_T Demand and Peak $\dot{V}_I$ Delivery on WOB_{sim}

Ventilator T_I Greater than Inspiratory Muscle Simulator T_I. WOB_{sim} rose substantially during VCV-CF and VCV-DF as V_T demand matched or exceeded the ventilator-delivered V_T (Fig. 2). In contrast, during PCV, WOB_{sim} rose less as V_T demand increased and generally was less than the corresponding WOB_{sim} measured during unassisted breathing. Peak flow demand exceeded ventilator peak $\dot{V}_I$ delivery only during VCV-CF at a V_T demand $\geq$ 100% (Fig. 3). During VCV-DF, ventilator peak $\dot{V}_I$ delivery always exceeded peak flow demand, but WOB_{sim} rose with increasing V_T demand (see Figs. 2 and 3). In contrast, during PCV the slope of increasing ventilator peak $\dot{V}_I$ delivery mirrored the slope of increasing peak flow demand during unassisted breathing (see Fig. 3). As V_T demand increased, WOB_{vent} fell precipitously to zero during VCV-CF and to approximately zero during VCV-DF (Fig. 4). During PCV, WOB_{vent} declined less (from 1.23 to 0.77 J/L). The sharp rise in WOB_{sim} seen at the highest level of V_T demand during VCV-CF and VCV-DF was attributed to high levels of WOB_{imp} from the ventilator's inspiratory valve (Fig. 5). During AMV with VCV-CF and VCV-DF, ventilator peak $\dot{V}_I$ and V_T delivery were tightly regulated, and they varied by only 2 L/min and 10 mL, respectively, from CMV conditions. In contrast, as flow and V_T demand rose during PCV, the ventilator-delivered peak $\dot{V}_I$ increased from 59 to 89 L/min and V_T increased from 400 to 688 mL.

Ventilator T_I Set at or below Inspiratory Muscle Simulator T_I. Decreasing the ventilator T_I so that it matched or was less than simulated T_I progressively increased ventilator peak $\dot{V}_I$ delivery, significantly reduced WOB_{sim} and WOB_{imp} during VCV-CF and VCV-DF (Tables 2 and 3). When V_T demand was 400–500 mL, the reductions in WOB_{sim} and WOB_{imp} during VCV approached that of PCV only when the ventilator T_I was 0.65 s (VCV-DF) and 0.45 s (VCV-CF). These reductions in T_I increased peak $\dot{V}_I$ delivery to 96 and 81 L/min, respectively, which matched or approached the corresponding peak $\dot{V}_I$ delivery observed during PCV (90–95 L/m). However, as T_I was reduced to 0.65 s during VCV-CF and VCV-DF, double-triggering occurred frequently at a V_T demand of 500

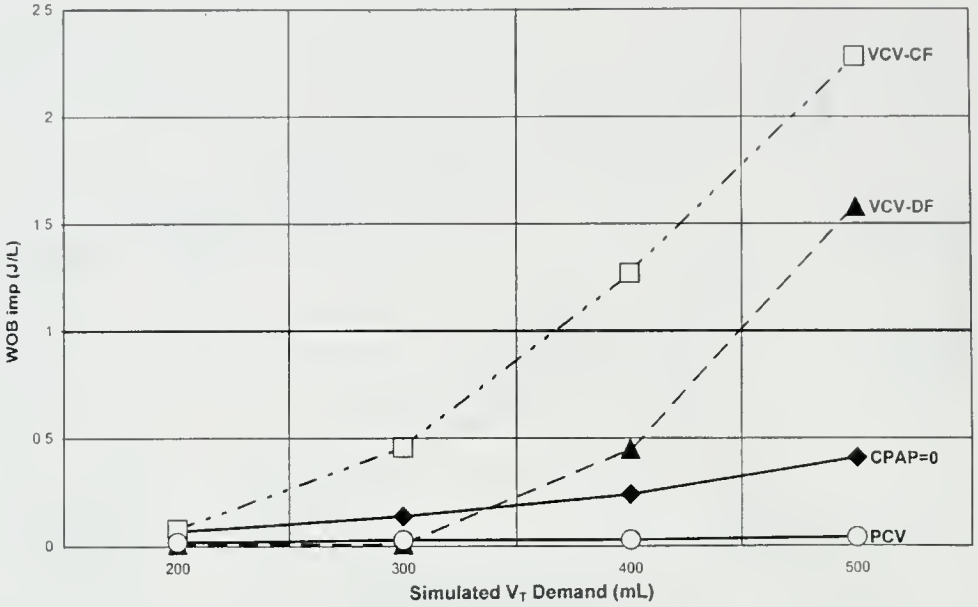

Fig. 5. Imposed work of breathing (WOB_{imp}) during assisted mechanical ventilation (inspiratory time 0.95 s) and zero continuous positive airway pressure (CPAP), as a function of tidal volume (V_T) demand (Protocol 1). The substantial increase in WOB_{imp} at 100% V_T demand during volume-control ventilation with constant flow pattern (VCV-CF) and volume-control ventilation with decelerating flow pattern (VCV-DF) paralleled the same changes in simulated work of breathing. WOB_{imp} during VCV exceeded the corresponding WOB_{imp} during unassisted breathing on CPAP.

mL, thus increasing the ventilator-delivered V_T from 400 to 800 mL. As T_I was reduced further (0.45 s) during VCV-CF, double-triggering occurred consistently at both 400 and 500 mL V_T demand (Fig. 6). When double-triggered breaths could be compared to single-triggered breaths at the same peak $\dot{V}_I$, there was a substantial further reduction in WOB_{sim} at the higher V_T delivery.

Decreasing ventilator T_I during PCV did not affect ventilator peak $\dot{V}_I$ delivery or WOB_{sim} but substantially decreased the amount of "overshoot" in V_T delivery. As T_I was reduced from 0.95 to 0.45 s (when V_T demand was 400 mL), the ventilator-delivered V_T decreased from 550 to 478 mL. When the same reduction in T_I was made at a V_T demand of 500 mL, the ventilator-delivered V_T decreased from 690 to 490 mL. Reducing T_I to 0.45 s also caused occasional double-triggering when V_T demand was $\geq$ 400 mL.

Use of Different Treatment Ventilators

When the high V_T demand (400–500 mL) portions of the protocol were repeated using different ventilators, similar magnitudes of WOB_{sim} and WOB_{imp} (and similar degrees of work reduction with T_I adjustments) were produced (Table 4). In contrast to the Veolar and Galileo ventilators, double-triggering did not occur with the Evita 2 dura ventilator. However, the ventilator-delivered V_T exceeded the preset value by 20% (483 mL). Scalar flow waveform analysis detected inspiratory flow generation during the end-inspiratory pause-hold, because continuing simulated effort opened the ventilator's internal antisuffocation valve.

Protocol 2: Constant Peak $\dot{V}_I$ Demand at Different Levels of V_T Demand

For VCV-CF and VCV-DF, WOB_{sim} and WOB_{imp} were substantially higher at 800 mL V_T demand than at 500 mL V_T demand, despite similar levels of peak flow demand (80 and 73 L/min, respectively) (Fig. 7). Although peak flow demand was inadvertently lower (9%) at the lower V_T demand, WOB_{sim} and WOB_{imp} were disproportionately reduced (64% and 68% less, respectively) at the lower V_T demand. The differences in WOB_{sim} could not be explained by differences in overcoming the elastic properties of the thoracic compartment at the different levels of V_T demand, because WOB_{sim} during unassisted breathing was within 10% (0.1 J/L). Thus, differences in WOB_{sim} could only be accounted for by differences in WOB_{imp} (see Fig. 7).

Discussion

We found that WOB_{sim} rose substantially during VCV-CF and VCV-DF when V_T demand matched or exceeded the ventilator V_T. This occurred even when venti-

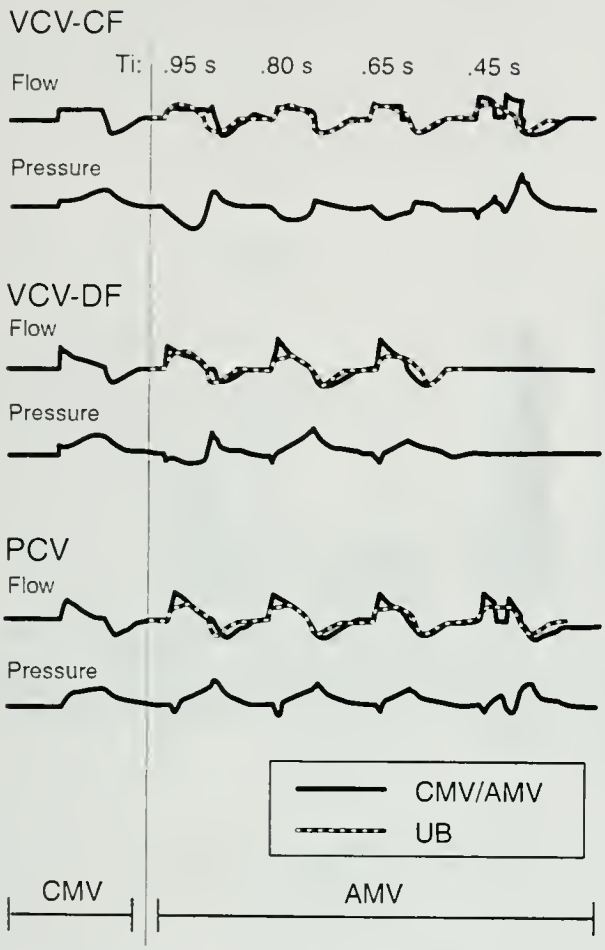

Fig. 6. The superimposition of flow demand waveforms upon flow delivery during assisted mechanical ventilation (AMV) (100% V_T demand) and the effects of ventilator inspiratory time (T_I) reduction on peak inspiratory flow ($\dot{V}_I$) and airway pressure. At T_I = 0.95 s peak flow demand exceeded ventilator $\dot{V}_I$ during volume-control ventilation (VCV). During VCV with decelerating flow pattern (VCV-DF), ventilator peak $\dot{V}_I$ occurred prior to peak flow demand. Decreasing T_I during VCV allowed better matching between ventilator peak $\dot{V}_I$ and flow demand. However, flow mismatching still occurred during the later portion of inspiration, as continued flow demand exceeded ventilator $\dot{V}_I$ delivery. When ventilator T_I was decreased to < 60% of simulated T_I (ie, T_I = 0.45 s), double-triggered breaths occurred during both VCV with constant flow pattern (VCV-CF) and pressure-control ventilation (PCV) (VCV-DF could not be modeled at this T_I setting.) During PCV, the peak $\dot{V}_I$ delivery and the flow waveform contour changed with simulated patient demand. CMV = controlled mechanical ventilation. UB = unassisted breathing.

lator peak $\dot{V}_I$ exceeded flow demand. When V_T demand exceeded the ventilator V_T, increasing ventilator peak $\dot{V}_I$ during VCV by decreasing T_I was of limited value in reducing WOB_{sim}. Substantially reducing ventilator T_I below simulated T_I during VCV consistently caused double-triggering, particularly when simulated V_T demand exceeded ventilator V_T delivery. By superimposing the scalar waveforms of flow demand with flow delivery, it was apparent that increasing ventilator peak $\dot{V}_I$ to treat V_T mismatching could not satisfy the overall flow-time requirements of the simulated patient (see Fig. 6). Thus, V_T mismatching during AMV can be thought of as mean $\dot{V}_I$ (V_T/T_I) mismatching.

When V_T mismatching occurred during VCV-CF and VCV-DF, increased WOB_{sim} was associated with rising levels of WOB_{imp}. Because WOB_{imp} was measured from the proximal end of the endotracheal tube (and no humidification device was used in the circuit), WOB_{imp} was caused by the ventilator's inspiratory valve attempting to maintain the operator-determined settings of $\dot{V}_I$ and V_T. The constancy of ventilator $\dot{V}_I$ and V_T delivery during volume-targeted AMV was maintained despite the inspiratory muscle simulator generating a P_{aw} approximately 3 times greater than was necessary (to produce the same volume and flow change) under CMV conditions. As the pressure gradient widens across the inspiratory valve, the ventilator can maintain a constant $\dot{V}_I$ target only by increasing valve resistance. Therefore, rather than "unloading" the inspiratory muscle simulator, the ventilator was in fact imposing "additional work" in a manner similar to that described in demand-flow continuous positive airway pressure systems.[17] WOB_{imp} was much greater during AMV when the ventilator was regulating flow to achieve a constant-volume target versus a constant-pressure target.

Although pressure-targeted modes have been used to deliver LPV,[10] there is concern whether the V_T target can be controlled adequately, particularly during AMV. As anticipated, excessive V_T delivery occurred during AMV and was partly responsible for maintaining WOB_{sim} at or below the levels found during simulated unassisted breathing. Reducing T_I from 0.95 to 0.45 s (when V_T demand was 400–500 mL) substantially diminished the degree of V_T "overshoot", from 2.2 to 4.3 mL/kg (above the 6 mL/kg target) to 1.3–1.5 mL/kg. Yet double-triggered breaths occurred occasionally. Thus, when patient-ventilator V_T mismatching results in frequent double-triggered breaths during VCV, PCV with a brief T_I may be a reasonable alternative, particularly when sedation cannot be increased further and neuromuscular blocking agents must be avoided.

There are several clinical implications arising from this experiment. First, the concept that mechanical ventilation *always* provides work assistance to the inspiratory muscles is not universally true. When V_T mismatching occurred during VCV, WOB_{vent} either approached or was zero. Second, our results do not support the assumption that total WOB remains more or less constant during VCV, with only the relative distribution of work between patient and ventilator altered by effort.[1] WOB_{imp} rose drastically during VCV and sometimes exceeded the WOB_{sim} needed to overcome the intrinsic elastic and resistive properties of the model. Third, the concept that flow asynchrony occurs when the ventilator fails to push the inspired gas at the

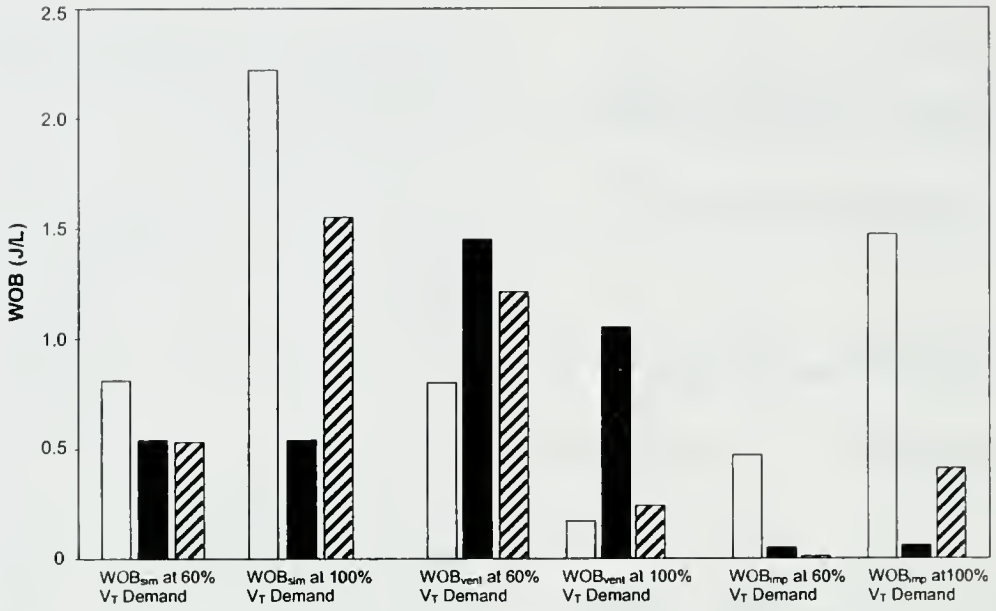

Fig. 7. Simulated work of breathing (WOB_{sim}), WOB performed by the ventilator (WOB_{vent}), and imposed WOB (WOB_{imp}) at similar levels of flow demand and different levels of tidal volume (V_T) demand (Protocol 2). During volume-control ventilation with constant flow pattern (white bars) and volume-control ventilation with decelerating flow pattern (hatched bars), both WOB_{sim} and WOB_{imp} were higher at 100% V_T demand than at 60% V_T demand, despite similar levels of peak flow demand (80 and 73 L/min). WOB_{sim} and WOB_{imp} were essentially the same during pressure-control ventilation (black bars) at both levels of V_T demand.

Table 2. Simulated Patient Work of Breathing As Ventilator Inspiratory Time Is Reduced*

V_T demand	Mode	Work of Breathing (J/L)			
		T_I = 0.95 s	T_I = 0.80 s	T_I = 0.65 s	T_I = 0.45 s
400 mL	VCV-CF	1.46	1.35	0.69	0.15†
	VCV-DF	0.83	0.69	0.17	—
	PCV	0.21	0.27	0.25	0.23†
500 mL	VCV-CF	2.55	2.24	0.78†	0.48†
	VCV-DF	1.93	1.37	0.32†	—
	PCV	0.34	0.46	0.38	0.32†

*At a constant simulated T_I of 0.8 s
V_T = tidal volume
T_I = inspiratory time
VCV-CF = volume-control ventilation with constant flow pattern
VCV-DF = volume-control ventilation with decelerating flow pattern
PCV = pressure-control ventilation
†Denotes presence of double-triggered breaths

same flow as the inspiratory muscles attempt to pull gas into the lungs[1,4] is inaccurate. We observed several instances when the ventilator peak $\dot{V}_I$ delivery matched peak flow demand during VCV-CF but WOB_{imp} was substantial ($\geq$ 0.45 J/L).

Furthermore, our model suggests that "unloading" the respiratory muscles during LPV may be particularly difficult to achieve, because pulmonary mechanics are abnormal. The compliance and resistance settings used in this model were within the range described for ARDS patients. Reducing WOB_{sim} even to a normal range (0.3–0.5 J/L)[27] in our model required that *both* ventilator peak $\dot{V}_I$ and V_T delivery greatly exceeded demand (by approximately 60–75% for peak $\dot{V}_I$ and 40–80% for V_T). Our 400 mL V_T target represented the average 6 mL/kg breaths (of a simulated 67 kg patient) from 2 LPV studies.[6,10] During PCV the delivered V_T that reduced WOB_{sim} to normal was 566–715 mL (8.4–10.7 mL/kg). Likewise, decreasing WOB_{sim} to normal during VCV required a very brief T_I that caused double-triggered breaths, resulting in a delivered V_T of 760–800 mL (11.3–11.9 mL/kg), which severely violated LPV goals. Hence our "push-pull" model of AMV suggests that complete unloading of the respiratory muscles may require the ventilator to displace the

Table 3. Imposed Work of Breathing As Ventilator Inspiratory Time Is Reduced*

V_T demand	Mode	Work of Breathing (J/L)			
		T_I = 0.95 s	T_I = 0.80 s	T_I = 0.65 s	T_I = 0.45 s
400 mL	VCV-CF	1.27	0.44	0.28	0.01†
	VCV-DF	0.45	0.27	0.01	—
	PCV	0.03	0.04	0.04	0.02†
500 mL	VCV-CF	2.28	1.94	0.56†	0.07†
	VCV-DF	1.58	0.91	0.10†	—
	PCV	0.04	0.05	0.04	0.03†

*At a constant simulated T_I of 0.8 s
V_T = tidal volume
T_I = inspiratory time
VCV-CF = volume-control ventilation with constant flow pattern
VCV-DF = volume-control ventilation with decelerating flow pattern
PCV = pressure-control ventilation
†Denotes presence of double-triggered breaths

Table 4. Simulated Patient Work of Breathing with 3 Ventilator Types*

Mode	Simulated Work of Breathing (J/L)			
	T_I (s)	Veolar	Galileo	Evita 2 dura
VCV-CF	0.95	1.46	1.40	1.11
	0.80	1.36	1.19	0.80
	0.65	0.69	0.64	0.69
	0.45	0.15†	0.15†	0.15‡
VCV-DF	0.95	0.83	0.62	—
	0.80	0.69	0.26	—
	0.65	0.17	0.10	—
	0.45	—	—	—
PCV	0.95	0.34	0.40	0.15
	0.80	0.28	0.22	0.15
	0.65	0.25	0.20	0.14
	0.45	0.23	0.20	0.07

*At 400 mL tidal volume demand. See text for descriptions of ventilators.
T_I = inspiratory time
VCV-CF = volume-control ventilation with constant flow pattern
VCV-DF = volume-control ventilation with decelerating flow pattern
PCV = pressure-control ventilation
V_T = tidal volume
†Double-triggering present
‡V_T overshoot (without double-triggering) present during VCV

chest faster, and perhaps to a greater degree than the target peak $\dot{V}_I$ and V_T intended by the patient.

Based on the dual goals of our experiment (minimizing WOB while maintaining a reasonable V_T target for LPV), we suggest that suspected V_T mismatching during VCV should be treated first by reducing T_I and incorporating an exponential decelerating flow pattern to increase peak $\dot{V}_I$. A trial of PCV should be considered if decreasing T_I fails to reduce patient effort or when double-triggered breaths become frequent. Using PCV with a short T_I may help limit V_T overshoot, although double-triggered breaths can occur. Despite these ventilator adjustments, LPV ultimately may require higher levels of sedation or more frequent use of neuromuscular blocking agents. In fact, a recent survey reported that patient-ventilator asynchrony and sedation issues were substantial barriers to maintaining LPV during the ARDS Network trial.[28]

Given the severe levels of WOB_{imp} found in this study, one may question the need for an LPV strategy when the patient can breathe spontaneously at a higher V_T and a substantially lower P_{aw}. In ARDS, excessive transpulmonary pressure is believed to cause ventilator-induced lung injury.[7] Thus, when partial ventilatory support modes were used in 2 LPV studies,[7,10] liberal amounts of sedation and neuromuscular blocking agents were used to ensure that V_T did not exceed 6 mL/kg.

The extension of our results to clinical practice is constrained by the limitations of our model. First, an analog for pleural pressure could not be represented. Thoracic compartment pressure equaled the distal endotracheal tube

pressure, so WOB_{sim} approximated total WOB_{imp} (inspiratory valve + endotracheal tube) rather than simulating "total patient" WOB. The lung parenchyma blunts P_{aw} transmission to the pleural surface. As lung compliance decreases and less P_{aw} is transmitted, positive-pressure ventilation becomes less effective in counteracting the development of negative muscle pressure and, thus, in reducing patient WOB.

Second, under conditions of flow and V_T mismatching, it is improbable that patient WOB would remain stable. It is more likely that a positive feedback loop would develop, because increasing respiratory muscle strain is sensed by the brain and typically increases respiratory motor neuron output to the breathing muscles.[29,30] This would tend to exacerbate patient-ventilator mismatching even more and further increase WOB. The value of our model is that it elegantly represents the "push-pull" concept of WOB during AMV[1] and thus allows testing of basic concepts about patient-ventilator interactions.

Third, our ventilators used PCV with pressure and flow algorithms that allowed for nearly instantaneous pressurization and peak $\dot{V}_I$ delivery. Some ventilators incorporate a pressure-rise time feature that allows the clinician to control the timing of inspiratory peak P_{aw}.[31] Pressure-rise time was not used during PCV, because it slows the rate-rise in $\dot{V}_I$ and increases WOB_{sim}.[32] In addition, we studied VCV-DF with an exponential decay pattern that increased peak $\dot{V}_I$ by 70% above the constant-flow waveform at the same T_I. Thus, our results would probably be reproduced only when pressure-rise time is not used during PCV, and in ventilators that incorporate a similar method for delivering a decelerating flow pattern in the VCV mode.

Conclusions

During VCV, simulated patient-ventilator V_T mismatching substantially increased WOB. This was directly attributed to WOB_{imp} by the ventilator's inspiratory valve, which was attempting to maintain operator-selected $\dot{V}_I$ and V_T values when there was excessive simulated demand. Therefore, the concept that mechanical ventilation *always* assists the patient by reducing WOB is not universally true. Our results suggest that V_T demand may exert an independent effect on WOB because of an intractable mismatching in the flow-time requirements between ventilator and patient when V_T delivery is constrained below patient demand. Therefore, increasing ventilator peak $\dot{V}_I$ to reduce patient WOB and improve comfort may have limited efficacy during LPV. Nevertheless, given these limitations, WOB_{imp} during LPV might be ameliorated somewhat by moderate reductions in T_I and by incorporating a decelerating flow waveform with an exponential decay. PCV with a brief T_I may effectively reduce WOB_{imp} but will probably cause a moderate violation of the V_T target. When T_I is too brief, both VCV and PCV result in double-triggered breaths that may increase the risk of ventilator-induced lung injury if the V_T repeatedly exceeds the target range.

REFERENCES

1. Marini JJ, Rodriguez M, Lamb V. The inspiratory workload of patient-initiated mechanical ventilation. Am Rev Respir Dis 1986; 134(5):902–909.
2. Marini JJ, Capps JS, Culver BH. The inspiratory work of breathing during assisted mechanical ventilation. Chest 1985;87(5):612–618.
3. Ward ME, Corbeil C, Gibbons W, Newman S, Macklem PT. Optimization of respiratory muscle relaxation during mechanical ventilation. Anesthesiology 1988;69(1):29–35.
4. Amato MBP, Barbas CSV, Bonassa J, Saldiva PHN, Zin WA, de Carvalho C. Volume-assured pressure support ventilation (VAPSV): a new approach for reducing muscle workload during acute respiratory failure. Chest 1992;102(4):1225–1234.
5. Derenne JPH, Macklem PT, Roussos CH. The respiratory muscles: mechanics, control, and pathophysiology. Am Rev Respir Dis 1978; 118(1):119–133.
6. Ventilation with lower tidal volumes as compared with traditional tidal volumes for acute lung injury and the acute respiratory distress syndrome. The Acute Respiratory Distress Syndrome Network. N Engl J Med 2000;342(18):1301–1308.
7. Hickling KG, Walsh J, Henderson S, Jackson R. Low mortality rate in adult respiratory distress syndrome using low-volume, pressure-limited ventilation with permissive hypercapnia: a prospective study. Crit Care Med 1994;22(10):1568–1578.
8. Brochard L, Roudot-Thoraval F, Roupie E, Delclaux C, Chastre J, Fernandez-Mondejar E, et al. Tidal volume reduction for prevention of ventilator-induced lung injury in acute respiratory distress syndrome. The Multicenter Trial Group on Tidal Volume Reduction in ARDS. Am J Respir Crit Care Med 1998;158(6):1831–1838.
9. Brower RG, Shanholtz CB, Fessler HE, Shade DM, White P Jr, Wiener CM, et al. Prospective, randomized, controlled clinical trial comparing traditional versus reduced tidal volume ventilation in acute respiratory distress syndrome patients. Crit Care Med 1999;27(8) :1492–1498.
10. Amato MBP, Barbas CSV, Medeiros DM, Magaldi RB, Schettino GP, Lorenzi-Filho GL, et al. Effect of a protective-ventilation strategy on mortality in the acute respiratory distress syndrome. N Engl J Med 1998;338(6):347–354.
11. Kallet RH, Campbell AR, Alonso JA, Morabito DJ, Mackersie RC. The effects of pressure control versus volume control assisted ventilation on patient work of breathing in acute lung injury and acute respiratory distress syndrome. Respir Care 2000;45(9):1085–1096.
12. Kallet RH, Alonso JA, Luce JM, Matthay MA. Exacerbation of acute pulmonary edema during assisted mechanical ventilation using a low-tidal volume, lung-protective ventilator strategy. Chest 1999; 116(6):1826–1832.
13. Cinella G, Conti G, Lofaso F, Lorino H, Harf A, Lemaire F, Brochard L. Effects of assisted ventilation on the work of breathing: volume-controlled versus pressure-controlled ventilation. Am J Respir Crit Care Med 1996;153(3):1025–1033.
14. Haas CF, Branson RD, Folk LM, Campbell RS, Wise CR, Davis K Jr, et al. Patient-determined inspiratory flow during assisted mechanical ventilation. Respir Care 1995;40(7):716–721.
15. MacIntyre NR, McConnell R, Cheng KCG, Sane A. Patient-ventilator flow dyssynchrony: flow-limited versus pressure-limited breaths. Crit Care Med 1997;25(10):1671–1677.
16. Tobin MJ, Jubran A, Laghi F. Patient-ventilator interaction. Am J Respir Crit Care Med 2001;163(5):1059–1063.

17. Katz JA, Kraemer RW, Gjerde GE. Inspiratory work and airway pressure with continuous positive airway pressure delivery systems. Chest 1985;88(4):519–526.
18. Auler JOC Jr, Saldiva PHN, Martins MA, Carvalho CRR, Negri EMN, Hoelz C, Zin WA. Flow and volume dependence of respiratory system mechanics during constant flow ventilation in normal subjects and in adult respiratory distress syndrome. Crit Care Med 1990;18(10):1080–1086.
19. Blanch PB, Banner MJ. A new respiratory monitor that enables accurate measurements of work of breathing: a validation study. Respir Care 1994;39(9):897–905.
20. Petros AJ, Lamond CT, Bennett D. The Bicore pulmonary monitor: a device to access the work of breathing while weaning from mechanical ventilation. Anaesthesia 1993;48(11):985–988.
21. Glantz SA. Primer of biostatistics. New York: McGraw-Hill; 1997: 92–96.
22. Brochard L, Harf A, Lorino H, Lemaire F. Inspiratory pressure support prevents diaphragmatic fatigue during weaning from mechanical ventilation. Am Rev Respir Dis 1989;139(2):513–521.
23. Van de Graaff WB, Gordey K, Domseif SE, Dries DJ, Kleinman BS, Kumar P, Mathru M. Pressure support: changes in ventilatory pattern and components on the work of breathing. Chest 1991;100(4):1082–1089.
24. Kreit JW, Capper MW, Eschenbacher WL. Patient work of breathing during pressure support and volume-cycled mechanical ventilation. Am J Respir Crit Care Med 1994;149(5):1085–1091.
25. Banner MJ, Kirby RR, Gabrielli A, Blanch PB, Layon AJ. Partial and totally unloading respiratory muscles based on real-time measurements of work of breathing. Chest 1994;106(6):1835–1842.
26. Grimm LG. Statistical applications for the behavioral sciences. New York: John Wiley & Sons; 1993:247–250.
27. Otis AB. The work of breathing. Physiol Rev 1954;34:449–458.
28. Rubenfeld GD, Cooper C, Carter G, Thompson T, Hudson L. Barriers to providing LPV to patients with acute lung injury and acute respiratory distress syndrome (ALI/ARDS) (abstract). Am J Respir Crit Care Med 2001;163(5):A766.
29. Killian KJ, Campbell EJM. Dyspnea. In: Roussos C, Macklem PT, editors. The thorax. Lung biology in health and disease series. Vol 29, Part B. New York: Marcel Dekker; 1985.
30. Cherniack NS, Milic-Emili J. Mechanical aspects of loaded breathing. In: Roussos C, Macklem PT, editors. The thorax. Lung biology in health and disease series. Vol 29, Part B. New York: Marcel Dekker; 1985.
31. Bonmarchand G, Chevron V, Menard J-F, Girault C, Moritz-Berthelot F, Pasquis P, Leroy J. Effects of pressure ramp slope on the work of breathing during pressure support ventilation in restrictive patients. Crit Care Med 1999;27(4):715–722.
32. Kallet RH, Alonso JA, Diaz M, Marks JD. Pressure-rise time increases work of breathing during assisted pressure control ventilation (abstract). Crit Care Med 1999;27(1):A94.

That Darn Scapula: A Common Pitfall in Interpreting the Chest Radiograph

Stanley G Cheng MD and Eric J Stern MD

Introduction
Scapula Anatomy
Lung Parenchymal Abnormality Mimics
Pleural Abnormality Mimics
Bone Pathology Mimics
Other Pitfalls
Summary

For the inexperienced interpreter of chest radiographs, the normal scapula can mimic some very common and potentially serious abnormalities. Failure to recognize this normal bony structure can lead to needless diagnostic work up and treatment. The potentially mimicked abnormalities fall into 3 major categories: lung parenchymal abnormalities, pleural abnormalities, and bone abnormalities. This report discusses scapula anatomy as it pertains to chest radiographic anatomy, and the reasons for misinterpretations, and shows examples of the scapula mimicking pathologic processes.
Key words: scapula, radiograph, radiography, x-ray, mimic, misdiagnosis. [Respir Care 2002;47(8):910–914]

Introduction

The scapula is an essential part of the shoulder girdle, being an attachment for the rotator cuff muscles and part of the shoulder joint itself. For the inexperienced reader of chest radiographs, the normal scapula can mimic some very common and potentially serious abnormalities. Failure to recognize this normal bony structure can lead to needless diagnostic work up and treatment. The potentially mimicked abnormalities include lung parenchymal abnormalities, pleural abnormalities, and bone abnormalities. This pictorial essay discusses scapular anatomy, especially as it pertains to chest radiographic anatomy and the reasons for misinterpretations, and shows examples of the scapula mimicking pathologic processes.

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Scapula Anatomy

The scapula is a thin, flat, triangular bone in the superior posterolateral thorax, composed of the body, neck, coracoid process, spine, and acromion. The body of the scapula is typically responsible for the majority of mimicked pathology. In a traditional posteroanterior chest radiograph the patient's hands are at the waist and the arms are internally rotated, so the scapula is normally rotated off of the projected area of the lungs (Fig. 1).[1] With the arms in the anatomical position (at the sides), the medial edge of the scapula projects over the midclavicular line and the body of the scapula overlies portions of ribs 2 through 7.[2] The scapula moves and rotates with the position of the arm.

Fig. 1. Normal posteroanterior chest radiograph with arrows outlining scapulae.

Improper radiographic positioning or inability to obtain proper positioning (such as in the intensive care unit setting or in certain disease states) will place the scapula in various degrees of projection over the thorax. When the scapula is in an unexpected position, opacity caused by the scapula may not readily be identified as scapular in origin. Rather, as the observer focuses on the search for lung pathology, misinterpretation can occur.

Lung Parenchymal Abnormality Mimics

The body of the scapula is extremely thin. The gross specimen is often nearly translucent. In the frontal projection the scapula body can add a hazy opacity rather than the well-defined opacity typical of other bones. Carefully inspecting the margins of the opacity, however, will usually show a sharp bony margin that corresponds to the scapula. If the finding remains questionable, a lateral radiograph will reveal whether there is actually lung parenchymal disease.

Diffuse opacity within a portion of the hemithorax, due to the overlying scapula, might be mistaken for pneumo-

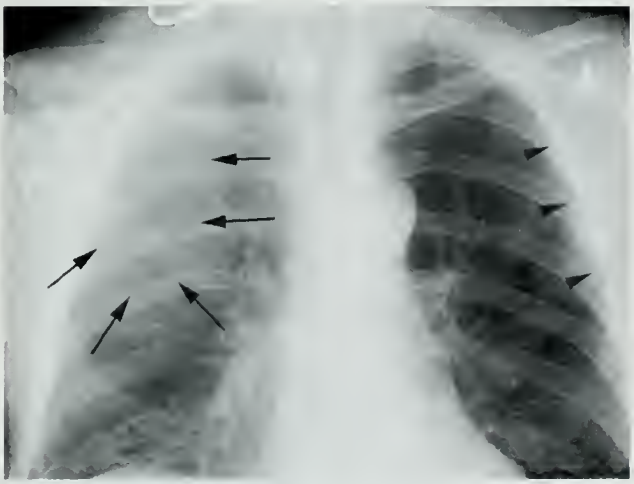

Fig. 2. Posteroanterior chest radiograph shows opacification of the right upper hemithorax due to the scapula (arrows), which might be mistaken for pneumonia. Arrowheads show the normal expected appearance of the left scapula. This finding occurs when the patient cannot raise the arm during the radiograph, thus not allowing the scapula to rotate out of the projected area of the lung.

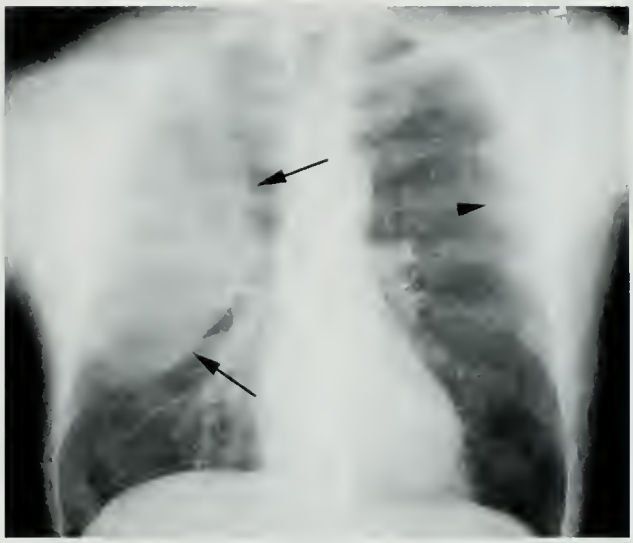

Fig. 3. Posteroanterior chest radiograph in a patient with winging of the scapula. A right upper lung opacity, which might be mistaken for pneumonia, is caused by the right scapula, in this case because it was not possible to properly position the scapula while taking the radiograph. The left scapula is normally positioned.

Fig. 4. Anteroposterior chest radiograph from the intensive care unit shows bilateral upper lung opacities that are due to overlying scapulae (arrows) and that could be mistaken for atelectasis. Right lower lung opacity is probably true subsegmental atelectasis. Arrowheads define the normal position of the minor fissure.

Fig. 5. Lordotic anteroposterior chest radiograph shows bilateral upper-lung linear opacities that suggest subsegmental atelectasis (arrows), but these opacities correspond to the spine of the scapula.

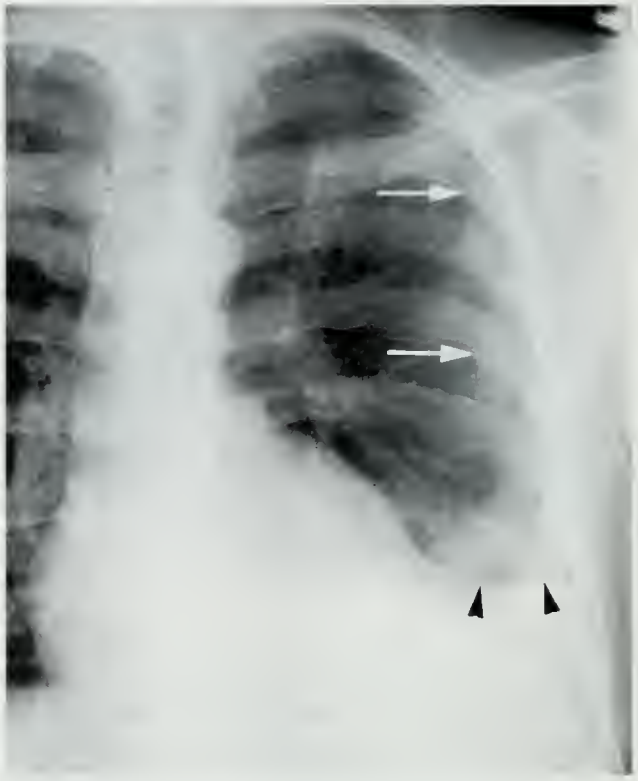

Fig. 7. An obvious, relatively small, left pleural effusion is present (arrowheads). There is also what appears to be separation of the visceral and parietal pleura (arrows), which is due to the scapula margin and that could lead one to conclude that the effusion is larger than it actually is or even that it is a loculated effusion.

Fig. 6. Posteroanterior chest radiograph shows what appears to be thickening of the pleura (arrows), but the opacity was actually caused by the medial scapula margin.

nia, especially if the patient has a clinical history of fever and cough (Fig. 2). On closer inspection the margins of the opacity will be seen to correspond to the typical contours of the scapula not rotated off of the hemithorax. If one of the scapulae is properly rotated off the hemithorax while the other is not, the asymmetry in density can also contribute to radiograph misinterpretation.

Certain disease states may not allow the technologist to properly position the patient's arms and scapulae. Winging of the scapula, caused by injury to the long thoracic nerve, causes paralysis of the serratus anterior muscle.[2] These patients will have difficulty abducting their arms, causing the scapulae to overlie the projected region of the lungs (Fig. 3).

Radiographs of patients in the intensive care unit frequently show various degrees of atelectasis. Bilateral upper lung opacities caused by overlying scapulae can easily be mistaken for atelectasis, with elevation of the right minor fissure or partial collapse of the left upper lobe, or both (Fig. 4). That conclusion might be reinforced by the presence of true lower-lobe atelectasis. Additionally, patient positioning for bedside radiography in the intensive care unit is highly variable, often leading to atypical scap-

Fig. 8. Anteroposterior chest radiograph shows bullet fragments (arrowhead) in the lower right chest and a right thoracostomy tube. The thin white line (arrow) in the right upper chest, which is due to the scapula, might lead one to conclude that right pneumothorax is present, especially given the bullet fragments and right thoracostomy tube. Note the bilateral symmetry of the scapular line as a clue to its real nature.

ular positioning that can cause radiographic opacities easily misinterpreted as lung pathology (Fig. 5).

Other potential lung parenchymal pathology misinterpretations caused by the scapula include nodules and masses. Bone islands within the scapula or a sclerotic scapular metastasis can be mistaken for lung masses. As is so often the case, a prior radiograph that shows the opacity in a different location with a different scapula position would help properly interpret the findings.

Fig. 9. What appears to be a small left pneumothorax (arrow) is caused by the Mach effect, secondary to the superior scapula margin.

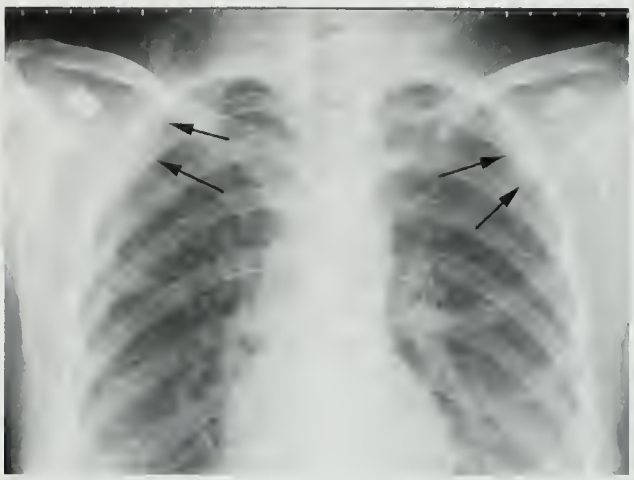

Fig. 10. Note what appear to be bilateral expansile second rib lesions (arrows), caused by the scapula overlapping ribs.

Pleural Abnormality Mimics

Even with proper radiographic positioning, occasionally the medial edge of the scapula can run parallel to the inner thoracic wall, creating an "edge" or thin white line that can be misinterpreted as pleural pathology.

What can appear to be thickening of the pleura can be caused by the medial edge of the scapula projecting adjacent to the lateral thorax (Fig. 6). To avoid this pitfall, follow the inferior margin of the apparent pleural thickening; it will be continuous with the tip of the scapula.

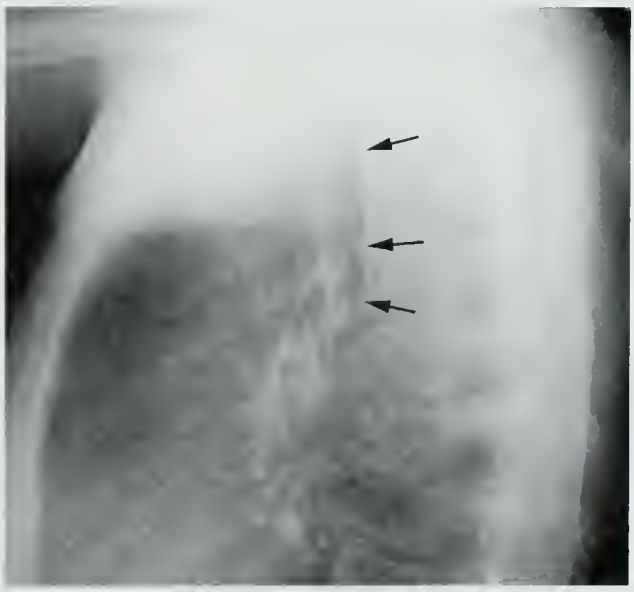

Fig. 11. Lateral chest radiograph shows what appears to be esophageal dilatation, but this is actually due to the scapula aligning behind the trachea (arrows). The anterior line is the posterior wall of the trachea.

If superimposed just right, the scapula margin can also lead one to conclude that an effusion is larger than it actually is, even suggesting loculation. Again, to avoid this pitfall, carefully follow the inferior margin of what appears to be a pleural effusion, and be sure to find the scapula (Fig. 7).

Diffuse opacification of the hemithorax on a supine radiograph suggests the presence of a large posteriorly layering pleural effusion. Most supine radiographs occur in the intensive care setting, where patient positioning is difficult and the scapula often overlies the projected area of the lungs. In a quick search for pathology the scapula might be mistaken for pleural effusion (Fig. 8).

The edge of the scapula can be mistaken for pneumothorax, especially if the clinical history is "line placement, rule out pneumothorax." Completely tracing what appears to be a pleural line will determine its scapular origin (Fig. 9).

Bone Pathology Mimics

When the scapula projects over the ribs or clavicle, bone pathology can be mimicked. Potential lesions include expansile or sclerotic bone lesions on the frontal chest radiograph and possibly ankylosis or diffuse idiopathic skeletal hyperostosis of the spine on the lateral view.

Confluence of overlapping rib shadows will often mimic rib pathology. Likewise, the scapula overlapping ribs may create this illusion. Again, by carefully tracing the borders of the scapula the scapular origin of the opacity will become clear (Fig. 10).

Other Pitfalls

The majority of misinterpretations will fall into one of the previously mentioned categories. However, the scapula can mimic other types of pathology as well, and the astute observer will always carefully consider whether this may be the case. For example, esophageal dilation can be mimicked on the lateral radiograph, when 2 parallel linear opacities project in the expected location of the esophagus (Fig. 11).

Summary

Unlike the other components of the chest radiograph, the scapula is unique in its mobility. It's position is highly dependent on many factors, so misinterpreting the scapula as intrathoracic pathology can be easy, especially when the observer is focused on determining whether acute lung pathology is present. If one looks carefully, however, one can avoid this pitfall by specifically looking for and tracing out the margins of the scapula. Keeping mindful of the scapula and its potential to mimic lung pathology will clarify questionable findings, especially when posteroanterior and lateral radiographs are viewed together. So remember, keep your eye out for that darn scapula.

REFERENCES

1. Ballinger PW. Merrill's atlas of radiographic positions and radiologic procedures. Volume one, 7th ed. St Louis: Mosby Year Book; 1991.
2. Moore KM. Clinically oriented anatomy. Baltimore: Williams & Wilkins; 1992.

Variable FEV_1 Following Methacholine Inhalation

Jeffrey M Haynes RRT RPFT

Introduction

A 72-year-old female nonsmoker was seen in the pulmonary function laboratory for spirometry and methacholine (Mch) bronchoprovocation challenge, because a diagnosis of asthma was being entertained as the source of her dyspnea on exertion. The patient had no history of pulmonary disease or smoking, denied recent chest infection, and was not taking bronchodilators or corticosteroids. Baseline spirometry values were normal, according to Knudson's 1983 reference values (Table 1).[1] Mch challenge was then begun, using a modified 5-breath dosimeter protocol, with Mch administered in accordance with American Thoracic Society guidelines.[2] Spirometry was repeated approximately 60 seconds after each dose of Mch, with all spirometry maneuvers occurring within 3 min of Mch inhalation. Figure 1 shows the changes in this patient's forced expiratory volume in the first second (FEV_1) in response to Mch inhalation.

Question

What are the possible causes of FEV_1 variability after individual Mch doses in this case?

Answers

1. Mch dissipation between spirometry maneuvers.
2. Inconsistent patient efforts.
3. Bronchodilation induced by deep inspiration (DI).

Discussion

Methacholine Dissipation

Care should be given to proceed through Mch challenge testing in a timely manner so that, to the best of your ability, consistent time intervals are achieved between Mch inhalations and spirometry measurements. American Thoracic Society guidelines recommend that all spirometry measurements be made within 3 min of Mch inhalation.[2] However, Mannino et al[3] examined the time course of spontaneous resolution of Mch-induced bronchoconstriction in asthmatics and found that statistically significant improvements in FEV_1 occurred after 30 min. Mch-induced bronchospasm may resolve quicker in patients with less sensitive airways.[4]

Inconsistent Patient Effort

Patient effort during spirometry testing may be inconsistent due to poor instruction, physical fatigue, compromised cognitive function, or indolence. Anyone trained to perform spirometry testing was no doubt taught that stern exhortations directing patient action during forced spirometry are very important to obtain maximum effort. The pulmonary function test technician must also try to demonstrate reproducibility by obtaining multiple measurements to validate consistent, presumably maximum efforts.[5] Indeed, one cannot overestimate the importance of a skilled technician with good patient assessment skills in obtaining quality pulmonary function data.[6,7] Though variable forced vital capacity, FEV_1, and peak expiratory flow values may signal inconsistent patient effort during routine spirometry testing, this conclusion may not be applicable during Mch challenge testing, because forced vital capacity, FEV_1, and peak expiratory flow can be variable because of poor effort, bronchoconstriction, or bronchodilation. American Thoracic Society guidelines offer a quality-control grading system for FEV_1 variability following Mch inhalation but also concede that more data are needed to better define FEV_1 variability standards during bronchoprovocation testing.[2] The advanced age of this patient may have been a risk factor for poor FEV_1 reproducibility.[7] Despite the fact that the variability of her baseline FEV_1 measurements was 1%, this elderly lady may have been susceptible to fatigue from the repeated forced expiratory maneuvers required by Mch challenge testing.

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Table 1. Baseline Spirometry Results

Test	Predicted	Measured	% of Predicted
FVC (L)	2.35	2.27	96
FEV_1* (L)	1.86	1.87	100
FEV_1/FVC	0.80	0.82	103
$FEF_{25-75\%}$ (L/s)	1.84	2.24	122
$FEF_{50\%}$ (L/s)	2.61	3.68	141
FEF_{max} (L/s)	4.93	5.06	103
TET (s)	NA	8.33	NA

Predicted = mean predicted values per Knudson et al[3]
FVC = forced vital capacity
FEV_1 = forced expiratory volume in the first second
*FEV_1 variability from 3 FVC measurements = 1%
FEV_1/FVC = ratio of FEV_1 to FVC
$FEF_{25-75\%}$ = forced expiratory flow during the middle half of the FVC
$FEF_{50\%}$ = forced expiratory flow at 50% of the FVC
FEF_{max} = maximum forced expiratory flow
TET = total expiratory time
NA = not applicable

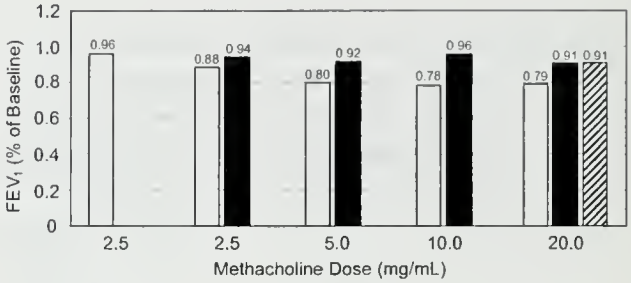

Fig. 1. The effect of methacholine inhalation on serial measurements of forced expiratory volume in the first second (FEV_1), expressed as a fraction of the baseline FEV_1. The white bars represent the first FEV_1 measurement after each dose of methacholine. The black bars represent the second FEV_1 measurement after each dose of methacholine. The hatched bar represents the third FEV_1 measurement after the 20 mg/mL dose of methacholine. The methacholine dose protocol was: 2.5 mg/mL (3 breaths); 2.5 mg/mL (5 breaths); 5 mg/mL (5 breaths); 10 mg/mL (5 breaths); 20 mg/mL (5 breaths).

Bronchodilation Induced by Deep Inspiration

Bronchoconstriction can be produced and maintained in nonasthmatics following Mch inhalation when DIs are prohibited.[8–13] Once DIs are performed, bronchodilation occurs. This phenomenon has been documented by both spirometry[8–13] and high-resolution computed tomography.[12] The similar airway response to Mch inhalation among asthmatics and nonasthmatics with DI prohibition led to the hypothesis that impaired DI bronchodilation might be responsible for asthmatic airway hyperresponsiveness;[8,13] however, Burns and Gibson[11] and Brusasco et al[10] showed that when both asthmatic and normal controls are challenged with Mch and prohibited DI, the asthmatics have greater decrements in lung function, which suggests a degree of hyperresponsiveness related to factors other than DI prohibition.

Fundamental respiratory physiology dictates that changes in parenchymal lung volume and airway caliber are related. Specifically, as alveolar volume increases during inspiration, airway resistance decreases and anatomical dead space increases. This relationship is the result of the tethering effect of the lung parenchyma on adjacent airways. This parenchyma-airway interdependence may limit the extent of airway smooth muscle (ASM) shortening, because the parenchyma represents a load against which ASM must contend. Factors that reduce parenchymal tethering, such as airway thickening, may thus enhance ASM contractility.[14] Loss of parenchyma-airway interdependence may be an important feature of nocturnal asthma. Irvin et al[15] demonstrated that the airway resistance (R_{aw}) amplification that occurs with lung volume loss in sleeping asthmatics is not reversed when lung volume is reestablished. In terms of the effect of lung volume on end-organ physiology as it relates to ASM contractility, an in vitro study of bovine tracheal smooth muscle suggested that tidal breathing and periodic sighs, which lengthen ASM, disrupt actin-myosin binding such that inspiration effectively curtails ASM contractile potential.[16] Therefore, preventing actin-myosin cycling in nonasthmatics by DI prohibition may facilitate methacholine-induced bronchospasm. It is unclear exactly why asthmatics have impaired DI bronchodilation (in fact, asthmatics may bronchoconstrict in response to DI).[17–19] Though some investigations have not found a correlation between markers of airway inflammation and impaired DI bronchodilation in asthmatics,[10,20] fluticasone has been shown to improve the bronchodilation response to DI in asthmatics, perhaps because of reduced peribronchial edema.[21]

Figure 1 shows that at Mch doses of 5 mg/mL, 10 mg/mL, and 20 mg/mL the initial FEV_1 was $\geq$ 20% below baseline, only to be followed by FEV_1 values 8%, 4%, and 9%, respectively, below baseline on subsequent forced expiratory maneuvers. The second FEV_1 measurement following each Mch dose occurs after the multiple DIs needed to construct flow-volume loops. Though it cannot be stated definitively that the lower FEV_1 values after each Mch dose were related to a lack of DIs during the period between Mch inhalation and FEV_1 measurement, since DI was not prospectively prohibited, it is difficult to dismiss the possibility that the DIs required for serial FEV_1 measurements amended Mch-induced bronchoconstriction.

Malmberg et al[22] studied the influence of FEV_1 measurement on FEV_1 values following Mch inhalation in nonasthmatics. When FEV_1 was measured immediately before Mch inhalation, post-Mch FEV_1 was higher than if FEV_1 was not measured before Mch inhalation. When FEV_1 was not measured immediately before Mch inhalation, the second and subsequent FEV_1 values after Mch inhalation were higher than the first.

Orehek et al[19] used inhaled carbachol to increase R_{aw} by 200% in asthmatics, then measured R_{aw} again after one DI. Once R_{aw} had returned to the pre-DI value (200% greater than baseline), FEV_1 was measured. FEV_1 decreased less in patients whose R_{aw} decreased after DI than in those whose R_{aw} increased following DI. The authors concluded that using FEV_1 to assess the bronchial response to pharmacologic challenge may be misleading, because DI can prevent changes in FEV_1. In patients who have variable FEV_1, specific airway conductance may serve as a less effort-dependent and lung-volume-sensitive end point.[2]

It is doubtful that the FEV_1 variability after Mch doses was due to Mch dissipation; as mentioned previously, all FEV_1 measurements were made within 3 min of Mch inhalation, and Mch-induced bronchoconstriction lasts approximately 30 min.[3] Whether the FEV_1 variability was due to inconsistent effort or DI-induced bronchodilation is subject to speculation and ultimately cannot be proven either way. It is my biased judgment (and I performed the pulmonary function testing) that the patient's effort was consistent. The FEV_1 variability in her baseline spirometry was 1%. After doses of Mch, FEV_1 declined significantly and returned to near baseline immediately following DIs taken prior to the second FEV_1 measurement. It is quite unlikely that a patient suffering from spirometry-related fatigue would have a larger FEV_1 on subsequent measurements; however, it could also be argued that perhaps initially poor efforts were recognized and improved upon for subsequent measurements by greater encouragement from the pulmonary function test technician. However, that scenario would have had to have been repeated 4 times. Because spirometry is an effort-dependent test, value reproducibility might be perceived as a specific gauge of patient effort; however, the potential of the spirometry maneuver to affect bronchial tone should also be considered as a source of FEV_1 variability.

REFERENCES

1. Knudson RJ, Lebowitz MD, Holberg CJ, Burrows B. Changes in the normal maximal expiratory flow-volume curve with growth and aging. Am Rev Respir Dis 1983;127(6):725–734.
2. Crapo RO, Casaburi R, Coates AL, Enright PL, Hankinson JL, Irvin CG, et al. Guidelines for methacholine and exercise challenge testing -1999. This official statement of the American Thoracic Society was adopted by the ATS Board of Directors, July 1999. Am J Respir Crit Care 2000;161(1):309–329.
3. Mannino F, Anticoli S, De Luca A, Terzano C. Time course of methacholine induced bronchoconstriction during drugs and spontaneous resolution. Allergol Immunopathol (Madr) 1996;24(2):70–74.
4. Mannino F, Anticoli S, Graziani E, Terzano C. [Study of spontaneous resolution of bronchial spasm after methacholine challenge: comparison of patients with different degree of hyperreactivity.] Recenti Prog Med 1997;88(3):115–119. *Article in Italian*
5. American Association for Respiratory Care. AARC Clinical Practice Guideline: Spirometry, 1996 update. Respir Care 1996;41(7)629–636.
6. Blonshine S. Integrating education with diagnostics: patient and technologist. Respir Care Clin N Am 1997;3(2):139–154.
7. Bellia V, Pistelli R, Catalano F, Antonelli-Incalzi R, Grassi V, Melillo G, et al. Quality control of spirometry in the elderly. The S.A.R.A. study. SAlute Respiration nell'Anziano = Respiratory Health in the Elderly. Am J Respir Crit Care 2000;161(4):1094–1100.
8. Fish JE, Ankin MG, Kelly JF, Peterman VI. Regulation of bronchomotor tone by lung inflation in asthmatic and nonasthmatic subjects. J Appl Physiol 1981;50(5):1079–1086.
9. King GG, Moore BJ, Seow CY, Pare PD. Time course of increased airway narrowing caused by inhibition of deep inspiration during methacholine challenge. Am J Respir Crit Care Med 1999;160(2):454–457.
10. Brusasco V, Crimi E, Barisione G, Spanevello A, Rodarte JR, Pellegrino R. Airway responsiveness to methacholine: effects of deep inhalations and airway inflammation. J Appl Physiol 1999;87(2):567–573.
11. Burns GP, Gibson GJ. Airway hyperresponsiveness in asthma: not just a problem of smooth muscle relaxation with inspiration. Am J Respir Crit Care Med 1998;158(1):203–206.
12. Brown RH, Croisille P, Mudge B, Diemer FB, Permutt S, Togias A. Airway narrowing in healthy humans inhaling methacholine without deep inspirations demonstrated by HRCT. Am J Respir Crit Care Med 2000;161(4 Pt 1):1256–1263.
13. Skloot G, Permutt S, Togias A. Airway hyperresponsiveness in asthma: a problem of limited smooth muscle relaxation with inspiration. J Clin Invest 1995;96(5):2393–2403.
14. Postma DS, Kerstjens HAM. Characteristics of airway hyperresponsiveness in asthma and chronic obstructive pulmonary disease. Am J Respir Crit Care Med 1998;158(5 Pt 3):S187–S192.
15. Irvin CG, Pak J, Martin RJ. Airway-parenchyma uncoupling in nocturnal asthma. Am J Respir Crit Care Med 2000;161(1):50–56.
16. Fredberg JJ, Inouye DS, Mijailovich SM, Butler JP. Perturbed equilibrium of myosin binding in airway smooth muscle and its implications in bronchospasm. Am J Respir Crit Care Med 1999;159(3):959–967.
17. Marthan R, Woolcock AJ. Is a myogenic response involved in deep inspiration-induced bronchoconstriction in asthmatics? Am Rev Respir Dis 1989;140(5):1354–1358.
18. Mitzner W, Brown RH. Potential mechanism of hyperresponive airways. Am J Respir Crit Care Med 2000;161(5):1619–1623.
19. Orehek J, Nicoli MM, Delpierre S, Beaupre A. Influence of the previous deep inspiration on the spirometric measurement of provoked bronchoconstriction in asthma. Am Rev Respir Dis 1981;123(3):269–272.
20. Pacini F, Filippelli M, Duranti R, Rosi E, Romagnoli I, Grazzini M, et al. Reduction in bronchodilation following a deep inhalation is poorly related to airway inflammation in asthma. Eur Respir J 1999; 14(5):1055–1060.
21. Corsico A, Pellegrino R, Zoia MC, Barbano L, Brusasco V, Cerveri I. Effects of inhaled steroids on methacholine-induced bronchoconstriction and gas trapping in mild asthma. Eur Respir J 2000;15(4):687–692.
22. Malmberg P, Larsson K, Sundblad BM, Zhiping W. Importance of the time interval between FEV_1 measurements in a methacholine provocation test. Eur Respir J 1993;6(5):680–686.

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Books, Films, Tapes, & Software

The Intensive Care Manual. Michael J Apostolakos MD and Peter J Papadakos MD. New York: McGraw-Hill. 2001. Soft cover, illustrated, 397 pages, $39.95.

As a clinician and educator in critical care medicine, I recognize the value of a concise text that presents the important topics and concepts pertinent to the intensive care unit (ICU). **The Intensive Care Manual** by Apostolakos and Papadakos addresses many of the common problems encountered in critical care medicine. From respiratory failure and mechanical ventilation to shock and renal failure, the authors follow an "approach"-based format that includes the important physiologic considerations that lend context to the pathophysiology. This is comparable to the "approach" format commonly used for bedside teaching by many clinician-educators.

Although the book is intended for "house officers, fellows, and attending staff," all members of the multidisciplinary critical care team will benefit from reading this manual. Clinical pharmacists, respiratory therapists, nutrition support specialists, and other clinical team members play an important role in managing ICU patients, so a common knowledge base and vocabulary are essential to the effectiveness of the team as a whole. **The Intensive Care Manual** can serve as a foundation for any clinician working in an ICU.

In the preface the authors describe their objectives for this manual. It is intended as a bedside reference for the trainee and staff, to facilitate diagnosis and management of problems commonly encountered in critically ill patients. Although they describe the content as being organized in organ-specific chapters, it is better described as organized according to major clinical issues within organ systems. Admittedly, this manual is not intended to be an exhaustive reference in critical care medicine. The usual comprehensive tome on critical care medicine is often so bogged down in minutiae that the novice clinician loses perspective on the key elements of diagnosis and management of the critically ill patient. This manual concentrates on a concise discussion of material relevant to intensive care. Although the order of the chapters does not necessarily follow the "head-to-toe" organ system approach, each chapter stands on its own. The authors are predominantly faculty from one academic center, but they represent disciplines with particular expertise in the topics presented. This is analogous to the consultation practice commonly employed in the management of complex patients.

This paperback manual is organized into chapters, with a content outline on the title page of each chapter. The illustrations, figures, and tables are effective in guiding the reader through the topics. Common formulae, with their variables concisely defined, also accompany the concepts. This is especially true in the chapters that address respiratory, cardiovascular, renal, and neurological physiology. The illustrations in the chapter on intravascular access and hemodynamic monitoring typify the anatomic structures and physiologic waveforms that are key to the effective placement and use of intravascular devices. The chapter on nutritional support is particularly representative of the important concepts and management issues in an often poorly managed aspect of overall patient care. The chapter on infectious disease addresses the multifactorial nature of suspected infection in critically ill patients, by focusing on the validity of the diagnostic techniques as well as the empirical and specific treatment options available. The most common sites and sources of ICU infection are discussed.

In all, this manual is easy to read and concise. It would complement the library of the novice and experienced clinician in the ICU.

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The Intensive Care Unit Manual. Paul N Lanken MD, Editor. C William Hanson III MD and Scott Manaker MD PhD, Associate Editors. Philadelphia: WB Saunders. 2001. Soft cover, illustrated, 1,147 pages, $59.95.

The editors of **The Intensive Care Unit Manual** wanted to produce a manual of critical care medicine that would be useful to house staff in medical, cardiac, and surgical intensive care units (ICUs). Their goal was to create a textbook that is comprehensive in scope, thus being valuable no matter what discipline of critical care one is practicing. At the same time they wanted to produce a concise, practical, and quick reference for busy on-call physicians. I thought that would be an impossible task, but I was pleasantly surprised to find that the editors came very close to achieving that lofty goal. In the process they produced a critical care textbook with several excellent features.

The book is organized into 98 chapters and 5 sections. The first 2 sections address general issues in the care of ICU patients. Chapters 1 through 9 review basic principles and practices of critical care, including acute respiratory failure, mechanical ventilation, weaning and extubation, hemodynamic monitoring, use of sedatives and paralytics, and issues regarding vascular access. Several chapters in this section are dedicated to the various forms of shock (cardiogenic, hypovolemic, and septic). The chapters in this section are written at a basic level—an advantage for busy medical students and physicians-in-training, who lack the time to review detailed discussions of physiologic concepts. Chapters 10–15 continue this theme by addressing issues of care that are generic to many critically ill patients. Topics such as general supportive care, nutrition, noninvasive monitoring, use of antibiotics and other drugs, and issues regarding nosocomial infection and drug interactions are presented in a manner that emphasizes both the important principles of care and the specifics of management.

The last 3 sections of the book are problem-oriented. Chapters 16–25 focus on problems commonly encountered in ICU patients with specific diseases or particular physiologies, such as human immunodeficiency virus disease, chronic renal failure, cancer, and chronic liver failure. Care of the maternal-fetal unit is also presented. The emphasis is on special concerns that arise in the ICU care of these patients because of their underlying conditions. This section also offers guidance regarding management of end-of-life issues, failure to wean, rehabilitation, and swallowing disorders.

The fourth section will probably be the most valuable to the busy physician on call. Chapters 36–48 review common complications that occur in critically ill patients. The

complications are organized as symptoms or laboratory abnormalities, and include issues such as fever, sudden dyspnea/hypoxia, airway management, arrhythmia, chest pain, barotrauma, oliguria, and electrolyte disorders. There are also discussions of gastrointestinal (diarrhea, ileus), neurologic (seizures, altered mental status, increased intracranial pressure), hemodynamic (hypertension and hypotension), and generic (skin rashes and ulcers, edema) complications. These chapters emphasize how to *immediately* evaluate and manage the problems. Several of the chapters include lists of clinical pearls or bullet points to highlight critical concerns—a very useful feature.

The final and longest section of the book is a more traditional system-based discussion of common disorders that require ICU admission. This section is organized according to medical, obstetric, surgical, and traumatic disorders. The medical illnesses are further organized by organ system. All major organ systems (cardiovascular, gastrointestinal, hematologic, infection defense, neurologic, pulmonary, renal/metabolic, endocrine) are included. There is also a chapter on environmentally-related disorders. The emphasis is on medical conditions, but the discussions of surgical illnesses (especially trauma) are satisfactory.

The multidisciplinary content of the book is reflected by the variety of its authors. There are associate editors from both the medical and surgical disciplines, as well as broad representation from both medical and surgical subspecialties. The book has 132 contributors, about 75% of whom have faculty appointments at the University of Pennsylvania; most of the remainder come from other major academic institutions. Nearly half are at the senior (associate or full professor) level.

The chapters are well written, concise, and easily readable. There is little fluff to the discussions; the book's comprehensive scope and concise approach mandate that every sentence be relevant to the clinical issue discussed. There is also extensive use of tables and treatment algorithms for both evaluation and treatment. The illustrations are clear and relevant to the subject material. As one might expect with so many different contributors, there is some variation in style and format among the chapters, but this does not affect the overall quality of the material. The book is well edited, with only an occasional typographical error. The index and table of contents are well organized and appropriately detailed, with the exception of the inclusion of *acute respiratory distress syndrome* under the general category of *obstetric* problems requiring ICU admission.

My major criticism of **The Intensive Care Unit Manual** is that trying to be everything for everyone and yet concise results in superficial discussions of some problems. For example, the discussion of acetaminophen overdose includes a very cursory discussion of how to use the Rumack-Matthew nomogram to direct therapy with N-acetylcysteine. There is little to no discussion of its application in situations in which the dose or timing of the acetaminophen overdose are unknown or in question. Similarly, the discussion of intracranial pressure monitoring in acetaminophen-induced fulminant hepatic failure does not include the risk/benefit controversies of monitoring in meaningful detail. To some degree the extensive use of tables and algorithms helps minimize this shortcoming.

There is also inadequate discussion of certain technical aspects of care. For example, the chapter regarding venous catheter placement includes a diagram of the venous anatomy but not the surface anatomic landmarks that guide catheter insertion.

The book is published as a paperback. Although the corners of the front and back covers became frayed over 6 months, the book demonstrated the durability expected of paperback construction. The binding was strong and remained intact. The book measures approximately 7 × 10 × 1.5 inches, and it weighs several pounds, precluding its use as a pocket reference.

The book is targeted primarily at clinicians-in-training who are rotating through services in medical, surgical, and coronary care units. It is weighted heavily toward the medical conditions that result in ICU admissions. However, the discussions of surgical diseases are adequate and the inclusion of the general principles of care and complications that occur in all ICU patients make this a valuable resource for surgical house officers as well. It will probably also be valuable as a quick reference for practicing hospitalists who are responsible for ICU patients. Because of the book's concise nature, it is of limited benefit to practicing intensivists and intensivists-in-training, who probably will be interested in more detailed discussions of the subject matter. The lack of "hands on" discussions of technical aspects of ICU care limits its value to nurses and respiratory therapists. However, providers who are interested in understanding the medical strategy behind management decisions would find it valuable.

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Ventilator Management Strategies for Critical Care. Nicholas S Hill and Mitchell M Levy, Editors. (Lung Biology in Health and Disease, Volume 158, Claude Lenfant, Executive Editor.) New York: Marcel Dekker, 2001. Hard cover, illustrated, 833 pages. $225.

Textbooks on mechanical ventilation generally focus on 5 topics: physics, physiologic effects, monitoring, complications, and description of ventilation modes. Mechanical ventilation strategies, when discussed at all, are typically relegated to chapter subheadings or scattered in paragraphs throughout the book. Thus, a well organized textbook offering a comprehensive approach to ventilation strategies has been lacking. This shortcoming is somewhat surprising, considering that mechanical ventilation has been used routinely for the past 40 years. With the publication of **Ventilator Management Strategies for Critical Care** we now have a textbook dedicated to the application of mechanical ventilation to specific clinical concerns.

This textbook is part of the excellent series Lung Biology in Health and Disease (Claude Lenfant, executive editor) and complements an earlier volume in the series, *Physiologic Basis of Ventilatory Support*. The editors of **Ventilator Management Strategies for Critical Care** are to be commended for producing a textbook that is both well conceived and well developed. The chapters are uniformly concise, well written, and easily accessible to all critical care practitioners familiar with mechanical ventilation.

The book contains 22 chapters divided into 6 parts. Part One deals with the demographics and epidemiology of acute respiratory failure and includes chapters discussing its 2 major forms (acute lung injury and chronic obstructive pulmonary disease), an overview chapter on the evolution of mechanical ventilation, and 2 chapters that describe how mechanical ventilation is used, both worldwide and in the United States.

The chapter on practice in the United States provides a sobering view of the present and future economic impact of mechanical ventilation. This provocative discussion casts a shadow that looms over the remainder of the book, and underscores the urgency of developing, testing, and implementing strategies that can positively impact patient outcomes.

Part Two, "Strategies for Optimal Utilization of Mechanical Ventilation in Critical Care," is the least cohesive section of the book, but the approach used for this part was necessary to address a diverse range of issues crucial to the development of subsequent topics. The section includes chapters on protocol-driven mechanical ventilation strategies, noninvasive mechanical ventilation, new modes of mechanical ventilation, closed-loop ventilation, treatment of agitation in the critical care setting, and optimal airway management.

Parts Three through Five contain the central topics. Part Three, "Ventilator Strategies for Specific Causes of Respiratory Failure," contains chapters updating management issues and techniques for patients with asthma, chronic obstructive pulmonary disease, and acute lung injury, and a chapter on noninvasive mechanical ventilation. In Part Four, "Strategies to Facilitate the Weaning Process," there are 3 chapters, which discuss, in turn, strategies for predicting successful weaning, optimizing the efficiency of weaning, and the early application of noninvasive ventilation as a weaning alternative. Part Five, "Strategies to Minimize Complications of Mechanical Ventilation," contains 3 brief chapters describing how to avoid ventilator-induced lung injury, the diagnosis and management of ventilator-associated pneumonia, and the effects of mechanical ventilation on heart-lung interactions.

The book concludes with a section on outcomes of mechanical ventilation, which contains only 2 chapters: one discussing extubation failure and the other describing ethical considerations involved in the withholding and withdrawal of mechanical ventilation.

The only criticism I have of this book (and it is minor) is the lack of a summation chapter discussing the overall "outcomes" of mechanical ventilation. Taking into account the stunning economic implications of mechanical ventilation practice, I think a modest amount of philosophical perspective (written either by the editors or an "elder statesmen" of mechanical ventilation) would have provided closure to the book.

I approached the review for this textbook in 2 ways. First I used it as a resource for issues on which I was not particularly well read. Second I read chapters on topics that I have researched. Regarding the first approach, the chapter on ventilator-associated pneumonia provided straightforward information on epidemiology, pathogenesis, and diagnostic techniques. This allowed me to quickly develop a pilot project for monitoring ventilator-associated pneumonia in my surgical intensive care unit. This initial impression was confirmed when I read the chapter on strategies for patients with acute lung injury. As an example, the issues surrounding recruitment maneuvers and pressure-volume curves are complicated and controversial. However, the authors wisely limit the discussion to only a few articles that highlight the major themes and boundaries of our current understanding. Thus the recent work by Pelosi et al[1] on the differing effects of recruitment maneuvers based on the distinctions between pulmonary and extrapulmonary forms of lung injury is discussed. The review of research by Ranieri[2] and Rimensberger[3] provides an overview of how the shape of pressure-volume curves relates to lung mechanics and disease stage and allows the reader to quickly grasp the complexities of pressure-volume curve interpretation. I found the same balance and parsimonious use of information in each chapter.

Ventilator Management Strategies for Critical Care is a hard-cover book with a sturdy, woven binding and high-grade paper. The chapters and tables are well designed and the illustrations are clear and easy to interpret. I found no typographical errors. Each chapter is richly referenced for further reading. This volume serves as a timely and valuable asset to clinical practice. It should be a welcome addition to anyone's medical library.

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REFERENCES

1. Pelosi P, Cadringher P, Bottino N, Panigada M, Carrieri F, Riva E, Lissoni A, Gattinoni L. Sigh in acute respiratory distress syndrome. Am J Respir Crit Care Med 1999;159(3):872–880.
2. Ranieri VM, Eissa NT, Corbeil C, Chasse M, Braidy J, Matar N, Milic-Emili J. Effects of positive end-expiratory pressure on alveolar recruitment and gas exchange in patients with the adult respiratory distress syndrome. Am Rev Respir Dis 1991;144(3 Pt 1):544–551.
3. Rimensberger PC, Pristine G, Mullen B, Cox PN, Slutsky AS. Lung recruitment during small tidal volume ventilation allows minimal positive end-expiratory pressure without augmenting lung injury. Crit Care Med 1999;27(9):1940–1945.

Practical Fibreoptic Intubation. Mansukh Popat MBBS. Oxford: Butterworth Heinemann. 2001. Soft cover, illustrated. 191 pages, $63.

Practical Fibreoptic Intubation, by Mansukh Popat, describes clearly and in great detail fiberoptic intubation techniques for managing the difficult airway—techniques that are challenging for many practitioners to master without some help. This practical soft-cover book (in a handy 8 × 5 inch size) has numerous beautiful color photos and illustrations. Directed toward anesthetists, intensivists, and emergency department physicians involved in airway management, this book will prove equally useful to those who teach fiberoptic skills (because the author outlines teaching methods) and those just learning the skills. The same specialists in pediatrics will find material directed toward them. The book will also be helpful to therapists and nurses who care for patients whose airways must be managed.

Mansukh Popat is a consultant anesthetist in Oxford, United Kingdom. His book is the first, to my knowledge, to be entirely devoted to fiberoptic intubation, since the second edition of Andranik Ovassapian's *Fiberoptic Airway Endoscopy in Anesthesia and Critical Care* (now titled *Fiberoptic Bronchoscopy and the Difficult Airway*) was published in 1996.

Popat's 191-page book is divided into 12 chapters. Chapters 1, 2, and 3 include a brief and predominately British history of fiberoptic developments and focus on fiberoptic equipment and developing basic fiberoptic training skills. For those wanting more practice there is a section on whether consent is required to do elective fiberoptic intubation on anesthetized patients. In Chapter 3 the author's short but outstanding description of basic endoscopy skills covers how to hold

the scope and manipulate its tip, the proper position of the endoscopist in relation to the patient, and especially the need to keep the shaft of the scope straight to accurately manipulate the tip. In my experience, many endoscopists appear not to understand the importance of those basic skills.

Subsequent chapters detail the step-by-step use of fiberoptic intubation in various kinds of difficult airways, in both anesthetized and awake patients, using nasal and oral approaches. There are sections on topical anesthesia of the airway and the use of the fiberoptic scope for endobronchial intubation.

The author could have emphasized 3 additional areas. First, in Chapter 4, in discussing the 12 fiberoptic airway aids he could have explained why there are so many types; their sheer number suggests that none is totally satisfactory. Second, he hardly mentions the tongue traction method, although it is useful for both awake and asleep patients in all sorts of difficult airway situations, even in children. In my 12 years of teaching fiberoptic intubation to anesthesia residents at the University of Washington, I've found that pulling the tongue forward is a more effective way to see the glottic opening through the fiberoptic scope than using any of the airway aids. Finally, Popat does not discuss the difficulties inherent in managing the airways of morbidly obese patients, in whom it is often preferable to do awake intubation because of their excess pharyngeal mucosa and low functional residual capacity.

The book has few errors. One error is in Figure 6.1, in which the nasal pharynx in photograph (e) is misidentified as the tongue. There are also a few typographical mistakes. On page 5 the Olympus LF-2 fiberscope is misidentified, and on page 73 reference 1 misspells Dr Fred Cheney's name. On page 134 some readers might be confused by the statement that a propofol infusion for sedation should be started "at 1 μg/mL", which is a concentration, not a rate.

Other than those minor caveats, this book belongs in the pocket of everyone involved in managing difficult airways. I highly recommend it and say "Two Thumbs Up!"

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Airway Management in the Critically Ill. Rade B Vukmir MD. New York: Parthenon Publishing Group. 2001. Hard cover, illustrated, 135 pages, $64.95.

The author's introduction states that this text is intended for novice, intermediate, and advanced practitioners, across a range of disciplines, including emergency medicine, anesthesia, and both medical and surgical critical care practitioners. The author has training and clinical experience across several disciplines: he is an administrator of an emergency department, was trained as an internist, and has credentials in both emergency medicine and critical care medicine.

The book is a thin, attractive hard-cover text divided into 5 chapters. The text in its entirety is only 135 pages, but each chapter includes extensive references. The references, chapter dividers, preface and index make up 42 pages, leaving only 93 pages of written text. The first chapter (a review of intubation and pharmacology) and the second chapter (on difficult intubation) are 28 and 29 pages, respectively, and therefore comprise more than half of the book. Chapter 3 covers confirmation of tracheal intubation and has only 4 pages of text. Chapter 4 has 12 pages of written material on laryngotracheal injury from prolonged intubation. The fifth and final chapter is on surgical airways, which are covered in 15 pages.

I noted the author's reference to Chevalier Jackson, the repeated mention of laryngoscopy in the preface, and the size of the first and second chapters relative to the rest of the text and therefore hoped that laryngoscopy would be covered in detail. Within the first few pages it became apparent that not only was this topic inadequately addressed, but the entire book and the manner in which it has been written, referenced, and assembled have substantial shortcomings. Direct laryngoscopy is the means of tracheal intubation in more than 99% of emergency airways, but in this text that technique is addressed in only 29 words, and those 29 words make up the labels that accompany 6 figures (Figures 4–9, page 13), beginning with "scissors mouth opening" and ending with an image of the "oropharynx and vocal cords." The figures begin with photographs of an adult, with the operator using a curved blade and opening the mouth without wearing gloves. The final image in this 6-figure sequence is a poor-quality line drawing of laryngoscopy on an infant, with a straight blade, in which neither the vocal cords nor any other laryngeal structures can be identified.

Other figures in the first 2 chapters also suffer numerous problems. A statement discussing a balanced approach to anesthesia, which mentions sedatives, muscle relaxants, and inhalational anesthesia, refers to a photograph (Figure 1) labeled "anesthesia work area." The next figure (Figure 2) is a photograph of an intubation manikin face, with its molded plastic mouth open and is labeled "laryngeal stimulus for blade insertion." The figure is referenced in a sentence that discusses mean arterial blood pressure and premature ventricular contractions during laryngoscopy in elective surgery cases. I have no idea what the author was intending to show or what that label means.

Other figures also display a remarkable lack of connection with the text, and the figures have little or no instructional value. For example, a sentence that discusses cricoid membrane puncture references a drawing of the external larynx and trachea (Figure 15) that does not show where the cricoid membrane is located. Instead it has labels to the superior and recurrent laryngeal nerves, even though the text does not discuss nerve blocks. Even worse is a photograph (Figure 17) labeled "catastrophic facial trauma," which shows a patient with no identifiable facial features, from a blast injury. The figure is referenced in a sentence that states that rapid-sequence intubation is used in the vast majority of trauma cases, but this is the prototypical case in which rapid-sequence intubation should be *avoided* and intubation should be accomplished with either an awake surgical approach or awake laryngoscopy. Using muscle relaxants in this case would be unimaginable given the inability to obtain a face seal to provide ventilation and the high likelihood of difficult laryngoscopy.

The book contains only 2 flow diagrams regarding decision-making. The first of these, which is in the first chapter, is labeled "airway management protocol." The second, in the second chapter, is a modified version of the American Society of Anesthesiologists' Difficult Airway Algorithm. In the "airway management protocol" (page 33) the flow diagram is poorly labeled and the diagram makes it appear that intubation *precedes* sedation and muscle relaxation. I surmised that the author intends "intubation" to mean "decision to intubate," but that is not explicitly stated, and the diagram's design does not make it clear. Assuming that is the case, the author proposes that intuba-

tion be attempted first with sedation, and only following a failed laryngoscopy should a muscle relaxant then be used. Though such a conservative approach may be appropriate in the anticipated difficult airway, routine use of this strategy leads to a lower first-pass success rate and trauma to the patient that can hinder subsequent laryngoscopy efforts. Awake laryngoscopy also increases the risk of gagging and emesis, in addition to causing substantial hemodynamic responses to laryngoscopy. It is contrary to the routine practice in most operating rooms, emergency departments, and major trauma centers, and no references or rationale are provided in support of this protocol.

The book has many important errors and omissions, along with outdated statistics, devices, and techniques. There is a discussion and 2 photographs of the esophageal obturator airway, even though that device is no longer available and all references to it have been removed from the Advanced Cardiac Life Support text and other reference texts (page 67). As in the photographs of mouth opening for laryngoscopy, the esophageal obturator airway operator is shown using no gloves when opening the mouth. There is even a photograph of digital intubation without gloves (Figure 48). There is no mention of the intubating laryngeal mask airway and, in fact, only 5 paragraphs and 1 photograph (Figure 57) of the laryngeal mask airway. There is no discussion whatsoever of mask ventilation, no techniques to optimize mask ventilation, and no references regarding the use of the laryngeal mask airway or Combitube (cuffed oropharyngeal airway) as rescue ventilation aids in the setting of failed rapid-sequence intubation. Although there are photographs of lighted stylets, the book makes no distinction between the Trachlight and simpler lighted stylets, and the 2 accompanying figures (Figures 50 and 51) do not show any discernable glow on the anterior neck. There is a picture (Figure 52) of an Endotrol directional control tracheal tube that is held in a way that the trigger cannot be pulled. The text cites surgical airway rates of 3–6% for failed rapid-sequence intubation in emergency settings, yet the references are to studies from 10 years ago or longer (pages 34 and 107). Recent data on the use of rapid-sequence intubation in emergency departments and air ambulances have surgical airway failure rates well below 1%. It also states that nasal intubation is used in 40% of emergency intubations, again referencing one 10-year-old study (page 107), even though numerous recent studies on emergency airway management describe its use in $\leq$ 1% of cases.

The chapter on surgical airways has no drawings or sequential photographs of either cricothyrotomy, tracheotomy, or retrograde wire intubation. In a discussion of preoperative airway evaluation the author recommends dynamic fluoroscopy (page 46), citing a 12-year-old study, which, although feasible, is not in common use and is particularly irrelevant for emergency airways. He states that fiberoptic devices are often used "as a last-ditch effort in the difficult airway" (page 60), when in fact such devices often work poorly following repetitive laryngoscopy, with resultant bleeding, edema, and clinical deterioration. In a discussion of Mallampati scores and Cormack and Lehane grades he confuses prediction of difficulty (ie, Mallampati classification) and resultant laryngeal view (Cormack-Lehane grading): "Mallampati and Cormack and Lehane scales have been validated as accurate predictors of intubation success," and "The Mallampati classification and Cormack grading systems when correlated are essentially equivalent, predicting a 10.4% and 11% incidence of difficult intubation, respectively."

Although the author seems to put particular emphasis on pharmacology, there are noticeable omissions and errors in this subject as well. There is no mention of the role of lidocaine in the setting of head injury; although the efficacy of this is debated it should have been addressed. In a review of induction agents, midazolam is stated to have a 1–2 hour duration of action (page 20). The author provides a confusing discussion on the safety of succinylcholine in the setting of burns and immobilization, concluding a paragraph with the statement, "succinylcholine should be avoided in those who are immobilized" (page 37). Although that statement follows a synopsis of a study of denervated muscle tissue, it should have been clarified, and the term "immobilization" is particularly unfortunate since it commonly refers to patients immobilized for cervical spine precautions.

The chapters are structured poorly and by the second chapter it is apparent that the same writing method is used for each section. Within a given section most paragraphs are essentially 1–3 sentence summaries of various individual studies, with minimal interpretation of the results. There is little in the way of coordinated arguments for a standardized approach to a given problem, and most sections ramble through various studies with poor continuity and without arriving at clear conclusions.

A careful look at the references throughout the text reveals an explanation for why so much of the material presented is outdated. In the first chapter there are 179 references, of which only 2 are within the last 6 years and only 24 are less than 10 years old. In the second chapter on the difficult airway, which has 178 references, no references predate 1996, 1 is from 1995, and only 32 are less than 10 years old. In the chapter on tube confirmation (Chapter 3) none of the 53 references are less than 10 years old. Throughout the text the author mentions "recent" studies, but in fact the studies cited are rarely recent. For example, in the case of end-tidal carbon dioxide detection devices (references 51–53), none of the studies are more recent than 1990. The reason there are photographs of procedures conducted without gloves is because these images were acquired before universal precautions became standard.

It is apparent the author compiled this text and reference collection some time ago, given the abrupt cessation of references after 1995. The figures, tables, and legends seem to have been handled as an afterthought. I find it hard to envision an educational role for this text, with any group of airway practitioners, at any level. It is very difficult to read, and although some of the clinical photographs are interesting, they correlate poorly with the written material. For the novice and intermediate practitioner this book will serve only to confuse. Although there may be some material of value to the more advanced practitioner it is minimal compared to the amount of outdated material and numerous mistakes and omissions.

I recommend other dedicated airway texts for those interested in the topic. For the novice, a great text (although out of print) is *Emergency Airway Management* by Gorback. For the advanced practitioner I recommend comprehensive texts such as Benumof's *Airway Management: Principles and Practice* or Norton's *Atlas of the Difficult Airway*. For more focused texts I recommend Ovassapian's *Fiberoptic Endoscopy and the Difficult Airway*, or Brimacombe, Brain, and Berry's *The Laryngeal Mask Airway: A Review and Practical Guide*.

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Respiratory Infections, 2nd edition. Michael S Niederman MD, George A Sarosi MD, and Jeffrey Glassroth MD, Editors. Philadelphia: Lippincott Williams & Wilkins. 2001. Hard cover, illustrated, 636 pages, $145.

Infections of the chest are a major part of everyday practice for most health care practitioners, especially those specializing in pulmonary medicine. From the simple upper respiratory tract infections that are so common to the rare and life-threatening infections such as Hantavirus, they create tremendous diagnostic challenges to the clinician and require great efforts on the part of respiratory therapists. To correctly diagnose and treat these disorders we need detailed understanding of pathogens, host defense mechanisms, diagnostic techniques, and therapeutic options, and the text **Respiratory Infections**, now in it's second edition, is one of the most concise and up-to-date accounts. The book brings a wealth of experience from clinicians and scientists involved in this field, and in a wonderfully written and nicely edited format.

The book is divided into 45 chapters and 6 parts. It starts appropriately with an in-depth overview of the pathogenesis of pneumonia. The first 9 chapters go through the details of various host defenses that protect the lung parenchyma, which is constantly exposed to the environment. This section starts with an overview of defense networks in lungs and goes through nonspecific mechanisms, such as cough and mucociliary clearance, to humoral factors, neutrophils, and cell-mediated immunity, and explores the ever-growing list of cytokine networks. There are chapters on host impairment associated with human immunodeficiency virus infection, development of antimicrobial resistance, and the interactions between the gastrointestinal tract and respiratory infections.

The book's second part deals with clinical syndromes of respiratory infections. This is a practice-based approach that would be very helpful to clinicians, because the chapters are organized by the variety of clinical syndromes that we diagnose and treat every day. From upper respiratory tract infections and acute bronchitis to exacerbation in chronic bronchitis, these chapters detail clinical manifestations, etiologies, and management. The chapters are somewhat arbitrary; there are 3 separate chapters on nosocomial infections, 1 on children, 1 on hospital-acquired pneumonia (in and out of the intensive care unit), and 1 on nosocomial pneumonia in surgical patients. The chapters "Pneumonia in the Elderly" and "Bronchiectasis" are particularly interesting.

Part III, which overlaps somewhat with Part II, discusses individual pathogens and groups of pathogens and provides very detailed discussions on the pathogens. Each pathogen and its history are described, with epidemiological aspects highlighted as needed. Most of the discussions are very interesting and almost all of them are clinically relevant.

Part IV discusses the methods used in diagnosing respiratory infections. Part V briefly discusses therapies. Part VI focuses on prevention of respiratory infections, both in the community and in the hospital.

The book is full of appropriate diagrams, graphs, radiographs, and pathology micrographs. Although there has been increasing regulation of what medical students and physicians can do at the hallway laboratory, color illustrations of common respiratory pathogens would have been helpful. Also, certain chapters have a lot of subject overlap.

Overall, this wonderfully written book is complete and an important addition to the bookshelf in any physician's office. It is well written for both beginners and the most experienced clinicians. No doubt it will be invaluable for pulmonologists. The subject matter is appropriate for most nurse practitioners and respiratory therapists.

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Asthma and Respiratory Infections. David P Skoner MD, editor. (Lung Biology in Health and Disease, Volume 154, Claude Lenfant, Executive Editor.) New York: Marcel Dekker. 2001. Hard cover, illustrated, 312 pages, $150.

Viral upper respiratory infections (URIs) rank among the most important causes of acute asthma episodes. This is especially so in children, in whom URIs are associated with the vast majority of asthma exacerbations. Yet, although that association is well known, there is little knowledge about how URIs worsen asthma. As a consequence, there have been few therapeutic options designed specifically to prevent or minimize the effects of URIs on asthmatics.

This book provides a fine summary of the current state of knowledge regarding URIs and asthma. The first section (Chapters 1–4) serves as an introduction and will be of interest to the general clinician as well as the subspecialist. The book begins with an overview of upper and lower respiratory infection agents (Chapter 1) and specifically discusses the viruses involved in asthma worsening (Chapter 2). Infection with respiratory syncytial virus and corona viruses is associated with wheezing in infants, some of which is related to small airway caliber in infants and may not persist into childhood. Although some studies suggest that early infection with these viruses may be a risk factor for subsequent asthma, this hypothesis remains unproven because of a selection bias inherent in those studies (Chapter 4). Because testing for respiratory syncytial virus is usually done on wheezing infants, it may be that the infection is uncovering a propensity to wheeze rather than being the cause of subsequent wheezing. As wheezing infants grow into childhood many of them stop wheezing but others develop persistent wheezing and asthma. Rhinovirus is the most important trigger for asthma exacerbations in these children, and Chapters 2 and 3 discuss the evidence for this association, the most convincing of which came about with the advent of polymerase chain reaction technology, which allowed sensitive measurement of viral products. This technology revealed that an astounding 80–90% of children with asthma exacerbations demonstrate concurrent viral upper respiratory infections.

Chapters 5–9 will be of interest primarily for asthma subspecialists and scientists who are concerned with asthma pathogenesis and mechanisms of disease. The general understanding of asthma (derived from allergen models) as primarily due to a genetic propensity for a Th2 (T helper cell type 2) response to airborne antigens is not applicable to URIs, which cause a Th1 response in both asthmatics and nonasthmatics. Therefore, new mechanistic hypotheses have been developed, some of which are discussed in Chapters 5 and 7. These include possible Th1-mediated eosinophilic inflammation and/or viral-induced dysfunction of the neural network that controls airway hyperactivity and tone.

A widely used approach to assess these hypotheses involves human challenge models, in which healthy and asthmatic subjects are infected experimentally (Chapters 6, 8, and 9). Unfortunately, the results of these small studies have been inconsistent. Some,

but not all, studies have found increased bronchial hyperreactivity. Some report higher cytokine production or cellular influx in asthmatics than in nonasthmatic controls. However, because of the small sample sizes and differences in experimental approach (eg, different strains of virus with potentially different pathogenicity), it is hard to know which results can be applied to the general asthma population with naturally occurring viruses. It is most intriguing that none of the challenge studies have been able to induce asthma exacerbations directly, although the doses administered are probably much higher than in natural transmission.

The third and final section of this book (Chapters 11–14) discusses diagnosis and treatment of URIs and URI-induced asthma exacerbations. These include over-the-counter medications, vaccines, and chemoprophylaxis with immune products such as interferon α. A short but timely introduction is presented of the ICAM-1 (intercellular adhesion molecule 1) inhibitors, which presently are the most promising potential therapy for rhinovirus-induced URIs. These chapters (except for the highly technical Chapter 12, which discusses polymerase chain reaction methodology) will be of interest to all clinicians.

This book is well-presented and generally well-written, although there is quite a bit of repetition from one chapter to the next. The tables and index are clear, the references are timely, and the information is for the most part accurate. I would dispute the conclusion in Chapter 13 that low-dose inhaled steroids are ineffective in the prevention of URI-induced exacerbations in kids. That conclusion was based on 1 study and is not supported by numerous other studies that have shown that low-dose inhaled steroids decrease childhood asthma exacerbations.

I would recommend this book as a thorough summary of a poorly understood but extremely important subject in childhood asthma.

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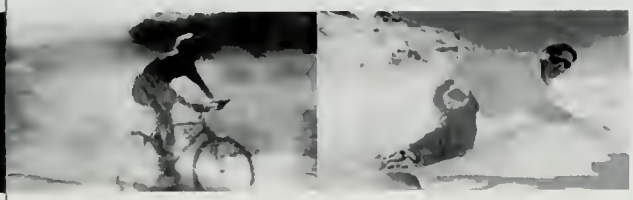
Denver, Colorado

The healing power of people.



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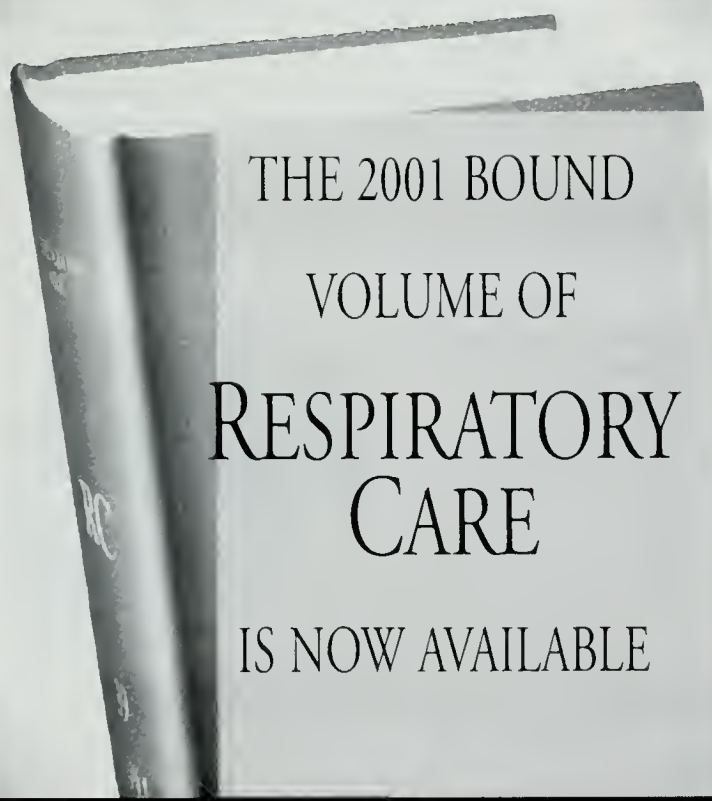

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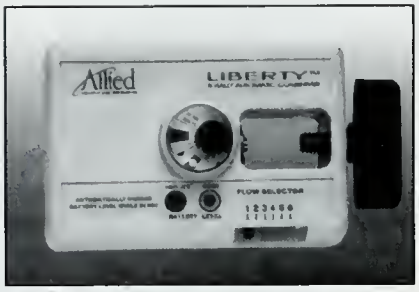
News releases about new products and services will be considered for publication in this section. There is no charge for these listings. Send descriptive release and glossy black and white photographs to RESPIRATORY CARE, New Products & Services Dept, 11030 Ables Lane, Dallas TX 75229-4593. The Reader Service Card can be found at the back of the Journal.

New Products & Services



Oxygen Conserver. Allied Healthcare Products has introduced its Liberty oxygen conserver. The features described by Allied Healthcare include an all-brass construction, which Allied says makes it both long lasting and compliant with NIOSH safety codes; further, it is totally automatic, engaging only when the patient starts to breathe and turning off when the cannula is removed from the patient's face. According to the manufacturer, a flashing green light on the display panel confirms that oxygen is being delivered and the unit is functioning properly. Other features named by Allied are that the unit weighs only 18oz, a red warning light flashes continuously from 24-48 hours when it's time for the battery (a single C battery) to be replaced, and it has a toggle switch that allows the patient to select the continuous flow mode. For more information from Allied Healthcare Products, circle number 184 on the reader service card in this issue, or send your request electronically via "Advertisers Online" at http://www.aarc.org/buyers_guide/

Sleep-Disordered Breathing Treatment Program. ResMed Corp has announced the enhancement of its SleepTrack™ program. In addition to patient outcomes management software, SleepTrack now includes materials to help ResMed's business partners improve their companies and grow, according to ResMed. The program consists of 4 modules—operations, marketing, education, and diagnostics—designed to increase a company's referral sources and optimize operational procedures, says the manufacturer. For more information from ResMed Corp, circle number 185 on the reader service card in this issue, or send your request electronically via "Advertisers Online" at http://www.aarc.org/buyers_guide/

Disinfectant Towels. Caltech Industries Inc has announced that their DISPATCH Hospital Cleaner Disinfectant Towels with Bleach now feature stronger and more durable towel fabric and come in convenient new packaging. These premoistened towels are disposable and are designed to clean and disinfect blood, body fluids, and other soil in one step, according to Caltech Industries. They are ideal, according to the manufacturer, for point-of-care equipment. Caltech also claims that the EPA-registered, fast-acting towels clean and disinfect for most bacteria and viruses in just one minute, and for TB in two minutes. They also meet OSHA, CDC, NCCLS, and Universal Precautions guidelines for bloodborne pathogen decontamination. For more information from Caltech Industries, circle number 186 on the reader service card in this issue, or send your request electronically via "Advertisers Online" at http://www.aarc.org/buyers_guide/

Oxygen Conserver. CHAD Therapeutics Inc has announced FDA clearance to market its new CYPRESS™ OXY Pneumatic® oxygen conserver. This is the first non-electric conserver offered by CHAD. According to the manufacturer, the CYPRESS conserver is a state-of-the-art pneumatic conserver that incorporates no electronic parts, thus eliminating the need for batteries. It is lightweight, small, and allows the use of a single-lumen cannula, says CHAD. It provides flowrates from 1-6 L/min and oxygen savings greater than 3:1 over continuous flow oxygen, according to the manufacturer. For more information from CHAD Therapeutics, circle number 187 on the reader service card in this issue, or send your request electronically via "Advertisers Online" at http://www.aarc.org/buyers_guide/

Notices

Notices of competitions, scholarships, fellowships, examination dates, new educational programs, and the like will be listed here free of charge. Items for the Notices section must reach the Journal 60 days before the desired month of publication (January 1 for the March issue, February 1 for the April issue, etc). Include all pertinent information and mail notices to RESPIRATORY CARE Notices Dept, 11030 Ables Lane, Dallas TX 75229-4593.

Scheduled Professor's Rounds 2002

Withholding and Withdrawing Life Support in the ICU — Gordon D Rubenfeld MDMSc / Richard D Branson BA RRT FAARC — Videotape Available

Weaning from Mechanical Ventilation: New Insights, New Guidelines — Neil R MacIntyre MD FAARC/ Dean R Hess PhD RRT FAARC — Videotape Available

Neonatal and Pediatric Ventilators: What's the Difference? — Mark J Heulitt MD FAARC/ Richard D Branson BA RRT FAARC — Videotape Available

Ventilator Graphs: What's With That Wave? — Jon O Nilsestuen PhD RRT FAARC/ Richard D Branson BA RRT FAARC — Videotape Available

Talking with Patients and Families About Death and Dying— Helen M Sorensen MA RRT FAARC/ David J Pierson MD FAARC —Live August 20; Audio September 17

Pressure vs Volume Ventilation: Does It Matter? — Robert S Campbell RRT FAARC/ Richard D Branson BA RRT FAARC — Live September 10/ Audio October 8

Inpatient Management of COPD — Randall Rosenblatt MD/ David J Pierson MD FAARC — Live October 22; Audio November 12

High-Frequency Oscillatory Ventilation — Thomas E Stewart MD/ Richard D Branson BA RRT FAARC — Live November 19; Audio December 10

Helpful Web Sites

American Association for Respiratory Care
http://www.aarc.org
— Current job listings
— American Respiratory Care Foundation fellowships, grants, & awards
— Clinical Practice Guidelines

National Board for Respiratory Care
http://www.nbrc.org

RESPIRATORY CARE online
http://www.rcjournal.com
— Subject and Author Indexes
— Contact the editorial staff
— OPEN FORUM; submit your abstract online

Asthma Management Model System
http://www.nhlbi.nih.gov

Keys to Professional Excellence
http://www.aarc.org/keys/

Committee on Accreditation for Respiratory Care
http://www.coarc.com

The National Board for Respiratory Care— Examination Fees for 2002

Examination	Examination Fees
CRT	$190 (new applicant) $150 (reapplicant)
Perinatal/Pediatric	$250 (new applicant) $220 (reapplicant)
CPFT	$200 (new applicant) $170 (reapplicant)
RPFT	$250 (new applicant) $220 (reapplicant)
RRT (Written & CSE)	$190 (new) $150 (reapplicant) written only $200 (new and reapplicant) CSE only $390 (new) $350 (reapplicant) both

For information about other services or fees, write to the National Board for Respiratory Care, 8310 Nieman Road, Lenexa KS 66214, or call (913) 599-4200, FAX (913) 541-0156, or e-mail: nbrc-info@nbrc.org

American Association for Respiratory Care

MEMBERSHIP APPLICATION

ACTIVE MEMBER

An individual is eligible if he/she lives in the U S or its territories or was an Active Member prior to moving outside its borders or territories, and meets ONE of the following criteria: (1) is legally credentialed as a respiratory care professional if employed in a state that mandates such, OR (2) is a graduate of an accredited educational program in respiratory care, OR (3) holds a credential issued by the NBRC.

ASSOCIATE OR SPECIAL MEMBER

Individuals who hold a position related to respiratory care but do not meet the requirements of Active Member shall be Associate Members. They have all the rights and benefits of the Association except to hold office, vote, or serve as chair of a standing committee. The following subclasses of Associate Membership are available: Foreign, Physician, and Industrial (individuals whose primary occupation is directly or indirectly devoted to the manufacture, sale, or distribution of respiratory care equipment or supplies). Special Members are those not working in a respiratory care-related field.

STUDENT MEMBER

Individuals will be classified as Student Members if they meet all the requirements for Associate Membership and are enrolled in an educational program in respiratory care accredited by, or in the process of seeking accreditation from, an AARC-recognized agency.

SPECIAL NOTICE — Student Members do not receive Continuing Respiratory Care Education (CRCE) transcripts. Upon completion of your respiratory care education, continuing education credits may be pursued upon your reclassification to Active or Associate Member.

Please read the eligibility requirements for each of the classifications above, then complete the form. All information requested must be provided, except where indicated as optional. See other side for more information and fee schedule. Please sign and date application on reverse side and type or print clearly. Processing of application takes approximately 15 days.

- ☐ Active
- Associate
  - ☐ Foreign
  - ☐ Physician
  - ☐ Industrial
- ☐ Special
- ☐ Student

Last Name _____

First Name _____

Social Security No. _____

Home Address _____

City _____

State _____ Zip __ __ __ __ __ - __ __ __ __

Phone No. (_____) _____

You are automatically assigned to a state society based on your home address. If you wish to be assigned to a different state society, please indicate which state that is here: _____

Work Information:

Place of Employment _____

Address _____

City _____

State _____ Zip __ __ __ __ __ - __ __ __ __

Phone No. (_____) _____

Preferred Fax No. (_____) _____

Preferred Email Address _____

Preferred mailing address: ☐ Home ☐ Business

Would you like to receive our monthly newsletter, AARC Report, by email?

☐ Yes ☐ No

Have you ever been or are you currently in the military?

☐ Yes ☐ No

Demographic Questions (optional)

We request that you answer these questions in order to help us design services and programs to meet your needs.

Primary Job Responsibility (check one only)

- ☐ Director (Technical or Program)
- ☐ Supervisor
- ☐ Diagnostic Technologist
- ☐ Instructor/Educator
- ☐ Other, specify _____
- ☐ Therapist/Technician
- ☐ Medical Director
- ☐ Student
- ☐ Staff Nurse

Type of Business

- ☐ Educational Institution
- ☐ DME/HME
- ☐ Home Health Agency
- ☐ Hospital/Acute Care
- ☐ Other, specify _____
- ☐ Manufacturer or supplier
- ☐ Outpatient Clinic
- ☐ Physician office
- ☐ Skilled Nursing Facility

Check the Highest Degree Earned

- ☐ High School
- ☐ RC Graduate Technician
- ☐ Associate Degree
- ☐ Bachelor's Degree
- ☐ Master's Degree
- ☐ Doctorate Degree

Number of Years in Respiratory Care

- ☐ 0-2 years
- ☐ 3-5 years
- ☐ 6-10 years
- ☐ 11-15 Years
- ☐ 16 years or more

Job Status

- ☐ Full Time
- ☐ Part Time

Credentials

- ☐ RRT
- ☐ CRT
- ☐ Physician
- ☐ CRNA
- ☐ RN
- ☐ LVN/LPN
- ☐ CPFT
- ☐ RPFT
- ☐ Perinatal/Pediatric

Date of Birth _____ Sex _____

FOR STUDENT MEMBER – REQUIRED

School/RC Program _____

Address _____

City _____

State _____ Zip __ __ __ __ __ - __ __ __ __

Phone No. (_____) _____

Expected Date of Graduation (REQUIRED INFORMATION)

Month _____ Year _____

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American Association for Respiratory Care **MEMBERSHIP APPLICATION**

Membership Fees

Payment must accompany your application to the AARC. Fees are for 12 months. These fees contain the $12.50 new members processing fee. Renewing members (except students) can deduct $12.50.

CHOOSE ONE LEVEL OF MEMBERSHIP

AARC REGULAR MEMBERSHIP (Receive both AARC Times and Respiratory Care journal)

☐ Active	$102.50
☐ Associate (Industrial or Physician)	$102.50
☐ Associate (Foreign)	$117.50
☐ Special	$102.50
☐ Student	$ 50.00

OR

AARC CHOICE MEMBERSHIP(Choose one publication)

☐ Active	$ 91.00
☐ Associate (Industrial or Physician)	$ 91.00
☐ Associate (Foreign)	$106.00
☐ Special	$ 91.00

I want ☐ AARC Times ☐ Respiratory Care journal

OR

AARC PLUS MEMBERSHIP (All publications and other items)

☐ Active	$137.50
☐ Associate (Industrial or Physician)	$137.50
☐ Associate (Foreign)	$177.50
☐ Special	$137.50

(Includes one free section - please mark choice below.)

Specialty Sections (optional)

Established to recognize the specialty areas of respiratory care, these sections publish a newsletter four times a year that focuses on issues of specific concern to that specialty. The sections also design the specialty programming at the national AARC meetings.

☐ Adult Acute Care Section	$15.00
☐ Education Section	$20.00
☐ Perinatal-Pediatric Section	$15.00
☐ Diagnostics Section	$15.00
☐ Continuing Care-Rehabilitation Section	$15.00
☐ Management Section	$20.00
☐ Transport Section	$15.00
☐ Home Care Section	$15.00
☐ Subacute Care Section	$15.00

PLEASE SIGN

I hereby apply for membership in the American Association for Respiratory Care and have enclosed my dues. If approved for membership in the AARC, I will abide by its bylaws and professional code of ethics. I authorize investigation of all statements contained herein and understand that misrepresentations or omissions of facts called for is cause for rejection or expulsion.

A yearly subscription to RESPIRATORY CARE journal and *AARC Times* magazine includes an allocation of $11.50 from my dues for each of these publications.

NOTE: Contributions or gifts to the AARC are not tax deductible as charitable contributions for income tax purposes. However, they may be tax deductible as ordinary and necessary business expenses subject to restrictions imposed as a result of association lobbying activities. The AARC estimates that the nondeductible portion of your dues — the portion which is allocable to lobbying — is 26%.

Signature _____

Date _____

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GRAND TOTAL = Membership Fee plus optional sections

$_____

☐ Total Amount Enclosed $_____

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Manuscript Preparation Guide

RESPIRATORY CARE welcomes original manuscripts related to the science and technology of respiratory care and prepared according to the following instructions and the *Uniform Requirements for Manuscripts Submitted to Biomedical Journals* (available at *http://www.icmje.org/index.html*). Manuscripts are blinded and reviewed by professionals with experience in the subject of the paper. Authors are responsible for obtaining written permission from the original copyright holder to use previously published figures and tables. Before publication, authors receive page proofs and are allowed to make only minor corrections. Accepted manuscripts are copy-edited for clarity, concision, and consistency with RESPIRATORY CARE's format. Published papers are copyrighted by Daedalus Inc and may not be published elsewhere without permission. Editorial consultation is available at any stage of planning or writing: contact the Editorial Office, 600 Ninth Avenue, Suite 702, Seattle WA 98104, (206) 223-0558, fax (206) 223-0563, E-mail: rcjournal@aarc.org

Categories of Articles

Research Article: A report of an original investigation (a study). Must include: Title Page, Abstract, Key Words, Introduction, Methods, Results, Discussion, Conclusions, and References. May also include: Tables, Figures (if so, must include Figure Legends), Acknowledgments, and Appendixes.

Review: A comprehensive, critical review of the literature and state-of-the-art summary of a topic that has been the subject of at least 40 published research articles. Must include: Title Page, Outline, Abstract, Key Words, Introduction, Review of the Literature, Summary, and References. May also include: Tables, Figures (if so, must include Figure Legends), Acknowledgments, and Appendixes.

Overview: A critical review of a pertinent topic that has fewer than 40 published research articles. Same structure as Review Article.

Update: A report of subsequent developments in a topic that has been critically reviewed in RESPIRATORY CARE or elsewhere. Same structure as a Review Article.

Special Article: A pertinent paper not fitting one of the other categories. Consult with the Editor before writing or submitting such a paper.

Editorial: A paper addressing an issue in the practice or administration of respiratory care. It may present an opposing opinion, clarify a position, or bring a problem into focus.

Letter: A brief, signed communication responding to an item published in RESPIRATORY CARE or about other pertinent topics. Tables, Figures, and References may be included. The letter should be marked "For Publication."

Case Report: Report of an uncommon clinical case or a new or improved method of management or treatment. A case-managing physician must either be an author or furnish a letter approving the manuscript. Must include: Title Page, Abstract, Key Words, Introduction, Case Summary, Discussion, and References. May also include: Tables, Figures (if so, must include Figure Legends), and Acknowledgments.

Point-of-View: A paper expressing personal but substantiated opinions on a pertinent topic. Must include: Title Page, Text, and References. May also include: Tables and Figures (if so, must include Figure Legends).

Drug Capsule: A miniature review paper about a drug or class of drugs. Drug Capsules address pharmacology, pharmacokinetics, and/or pharmacotherapy.

Graphics Corner: A brief, instructive case report discussing and illustrating waveforms for monitoring or diagnosis. Must include: Questions, Answers, and Discussion.

PFT Corner: A brief, instructive case report arising from pulmonary function testing, accompanied by a review of the relevant physiology and appropriate references to the literature. Must include: Questions, Answers, and Discussion.

Test Your Radiologic Skill: A brief, instructive case report pertinent to respiratory care and involving imaging, including one or more radiographs or other images submitted as black and white glossy photographs that clearly illustrate the teaching points being made. Must include: Questions, Answers, and Discussion.

Preparing the Manuscript

Double-space the text and number the pages. Do *not* include author names, author institutional affiliations, or allusions to institutional affiliations anywhere except on the title page. On the Abstract page include the title but do not include author names. Begin each of the following on a new page: Title Page, Abstract, Text, Acknowledgments, References, each Table, each Figure, and each Appendix. Use standard English in the first person and active voice. Type all headings in initial-capital letters (eg, Introduction, Methods, Patients, Equipment, Statistical Analysis, Results, Discussion). Center the main section headings and place second-level headings on the left margin.

Abstract. Please ensure that the abstract does not contain any facts or conclusions that do not also appear in the body text. Limit the abstract to no more than 250 words.

Key Words. Include a list of 6 to 10 key words or key phrases in Research Articles, Reviews, Overviews, Special Articles, and Case Reports. Key words are best selected from the Medical Subject Headings (MeSH) used by MEDLINE and available at *http://www.nlm.nih.gov/mesh/meshhome.html*.

References. Assign reference numbers in the order that articles are cited in your manuscript. At the end of the manuscript, list the cited works in numerical order. Abbreviate journal names as in *Index Medicus*. List all authors. If the research has not yet been accepted for publication, cite the research as a personal communication (eg, Smith KR, personal communication, 2001); *however, you must obtain written permission from the author to cite his or her unpublished data.* Do not number such references; instead, make parenthetical reference in the body text of your manuscript. Example: "Recently, Jones et al found this treatment effective in 45 of 83 patients (Jones HI, personal communication, 2000)."

The following examples show RESPIRATORY CARE's style for references.

Paper accepted but not yet published:

Hess D. New therapies for asthma. Respir Care (year, in press).

Article in a journal carrying pagination throughout the volume:

Legere BM, Kavuru MS. Pulmonary function in obesity. Respir Care 2000;45(8):967-968.

Article in a publication that numbers each issue beginning with Page 1:

Kallstrom TJ. Focus on asthma—disease management: a role for the respiratory therapist. AARC Times 1999;23(Oct):16, 17, 19.

Corporate author journal article:

American Association for Respiratory Care. Clinical Practice Guideline. Removal of the endotracheal tube. Respir Care 1999;44(1):85-90.

Article in journal supplement: (Journals differ in numbering and identifying supplements. Supply information sufficient to allow retrieval.)

Barnes PJ. Endogenous inhibitory mechanisms in asthma. Am J Respir Crit Care Med 2000; 161(3 Pt 2):S176-S181.

Abstract in journal: (Abstracts citations are to be avoided, and those more than 3 years old should not be cited.)

Volsko TA, De Fiore J, Chatburn RL. Acapella vs flutter: performance comparison (abstract). Respir Care 2000;45(8):991.

Editorial in a journal:

Giordano SP. What's that sound? (editorial) Respir Care 2000;45(10):1167-1168.

Editorial with no author given:

The perils of paediatric research (editorial). Lancet 1999;353(9154):685.

Letter in journal:

Piper SD. Testing conditions for nebulizers (letter). Respir Care 2000;45(8):971.

Book: (For any book, specific pages should be cited whenever reference is made to specific statements or other content.)

Cairo JM, Pilbeam SP. Mosby's respiratory care equipment, 6th ed. St Louis: Mosby; 1999:76-85.

Corporate author book:

American Medical Association Department of Drugs. AMA drug evaluations, 3rd ed. Littleton CO: Publishing Sciences Group; 1977.

Chapter in book with editor(s):

Isono S. Upper airway muscle function during sleep. In: Loughlin GM, Carroll JL, Marcus CL, editors. Sleep and breathing in children: a developmental approach. (Lung Biology in Health and Disease, Vol 147, Claude Lenfant, Executive Editor.) New York/Basel: Marcel Dekker; 2000:261-291.

World Wide Web

American Lung Association. Trends in pneumonia, influenza, and acute respiratory conditions mortality and morbidity. February, 2000. http://www.lungusa.org/data. Accessed November 20, 2000.

Tables. Tables should be consecutively numbered. At the bottom of the table define and/or explain all abbreviations and symbols used in the table. For footnotes use the following symbols, superscripted, in the table body, in the following order: *, †, ‡, §, ‖, ¶, **, ††. If data include a "±" value, please indicate whether the value is a standard deviation or standard error of the mean.

Figures (illustrations). Figures include graphs, line drawings, photographs, and radiographs. All figures should be sharp black-and-white images and be camera-ready. Glossy prints are preferred, but a good laser print will do. Use only illustrations that clarify and augment the text. Radiographs should clearly illustrate the point being made and should be submitted as black-and-white glossy photographs. If color is essential to the figure, consult the Editorial Office for more information. In reports of animal experiments, use schematic drawings, not photographs. A letter of consent must accompany any photograph of an identifiable person. Number figures consecutively as Figure 1, Figure 2, etc. All the figures must be mentioned in the text. Every figure must have a legend (a title and/or description explaining the figure). Figure legends should appear as separate paragraphs at the end of the manuscript (after the References section), in the same computer file as the manuscript (not in a separate file, as with the tables and figures).

Do not create scanned versions of figures borrowed from other publications; clear photocopies are preferable. To include figures previously published in other publications you must obtain permission from the original copyright holder. Figures must be of professional quality and a copy of the article from which the figure came should be available.

Drugs. Precisely identify all drugs and chemicals used, giving generic (nonproprietary) names, doses, and methods of administration. Brand or trade names may be given in parentheses after generic names.

Commercial Products. In the text, parenthetically identify commercial products only on first mention, giving the manufacturer's name and location. Example: "We performed spirometry (1085 System, Medical Graphics, Minneapolis, Minnesota)." Provide model

numbers if available, and manufacturer's suggested price if the study has cost implications.

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Ethics. When reporting experiments on human subjects, indicate that procedures were conducted in accordance with the ethical standards of the *World Medical Association Declaration of Helsinki* (see Respir Care 1997;42(6):635-636; also available at *http://www.wma.net/e/17-c_eparagraphnumbering.html*) or of the institution's committee on human experimentation. State that informed consent was obtained. Do not use patients' names, initials, or hospital numbers in text or illustrations. When reporting experiments on animals, indicate that the institution's policy, a national guideline, or a law on the care and use of laboratory animals was followed.

Statistics. Identify the statistical tests used in analyzing the data and give the prospectively determined level of significance in the Methods section. Report p values in the Results section. Cite only textbook and published article references to support choices of tests. Parenthetically identify any computer programs used. If data include a "±" value, please indicate whether the value is a standard deviation or standard error of the mean.

Units of Measurement. Express all measurements in SI (*Système Internationale*) units (units and conversion factors listed at Respir Care 1997;42(6):640 and also available at *http://www.rcjournal.com/author_guide/*. Show gas pressures (including blood gas tensions) in millimeters of mercury (mm Hg).

Conflict of Interest. On the cover page, authors must disclose any liaison or financial arrangement they have with a manufacturer or distributor whose product is addressed in the manuscript or with the manufacturer or distributor of a competing product. Such arrangements do not disqualify a paper from consideration and are not disclosed to reviewers. Reviewers are screened for possible conflict of interest.

Abbreviations and Symbols. Use the standard abbreviations and symbols listed at Respir Care 1997;42(6):637-642 (also available at *http://www.rcjournal.com/author_guide/*). Do not create new abbreviations. Do not use abbreviations in the title or section headings and do not use unusual abbreviations in the abstract. Use an abbreviation only if the term occurs 4 or more times in the paper. Parenthetically define *all* abbreviations: write out the full term on first mention, followed by the abbreviation in parentheses. Example: chronic obstructive pulmonary disease (COPD). Thereafter use only the abbreviation. Standard units of measurement and scientific terms can be abbreviated without explanation (eg, L/min, mm Hg, pH, O_2).

Please use the following forms: cm H_2O (not cmH2O), f (not bpm), L (not l), L/min (not LPM, l/min, or lpm), mL (not ml), mm Hg (not mmHg), pH (not Ph or PH), $p > 0.001$ (not $p>0.001$), s (not sec), S_{pO2} (arterial oxygen saturation measured via pulse-oximetry).

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Calendar of Events

Date	AARC & State Society Programs	Contact
Aug. 13	Professor's Rounds 2002 Teleconference, Ventilator Graphs: What's with That Wave?	AARC, (972) 243-2272
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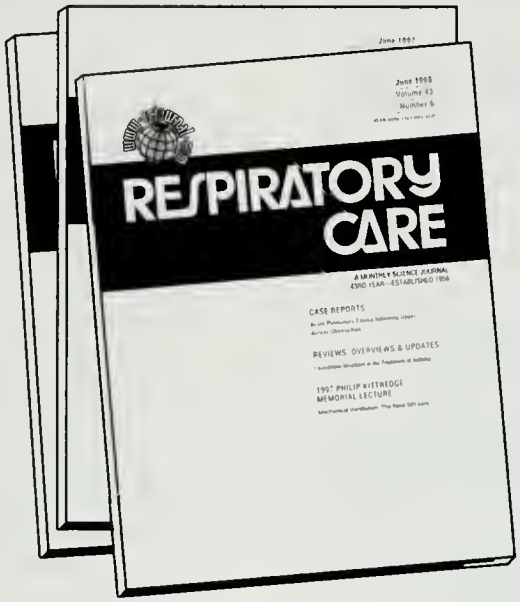
Sept. 5–7	TSRC North Region – Pineywoods District 16th Annual Fall Seminar; **Lufkin, TX**	Pam Hollis, (409) 639-7006
Sept. 10	Professor's Rounds 2002 Live Videoconference, Pressure versus Volume Ventilation: Does It Matter?	AARC, (972) 243-2272
Sept. 11–12	MSRC 25th Annual State Conference; **Sturbridge, MA**	Frances Smith (508) 833-9893, fsmith91@hotmail.com
Sept. 12–13	PSRC Southwest District's 29th Annual Seminar in Pulmonary and Sleep Medicine; **Pittsburgh, PA**	Gail Varcelotti (724) 941-8792, SWDistrict@psrc.net
Sept. 12–13	Alabama Society for Respiratory Care Annual Meeting; **Birmingham, AL**	Bill Pruitt (251) 434-3405 or wpruitt@jaguar1.usouthal.edu
Sept. 17	Professor's Rounds 2002 Teleconference, Talking with Patients and Families about Death and Dying	AARC, (972) 243-2272
Sept. 19–20	Kansas Respiratory Care Society Western Kansas Conference; **Hays, KS**	www.krcs.org or e-mail Julia Downs at jddrrt@yahoo.com
Oct. 5–8	AARC 48th International Respiratory Congress; **Tampa, FL**	AARC, (972) 243-2272, www.aarc.org

Date	Other Meetings	Contact
Sept. 27–29	13th Annual ELSO Meeting; Hilton Scottsdale Resort & Villas	Phoebe Hankins, (734) 998-6600, fax (734) 998-6602, phankins@umich.edu
Oct. 23	Spirometry Update and Refresher, **Cincinnati, OH**	Dr. Roy McKay, University of Cincinnati Medical Center, (513) 558-1234, www.DrMcKay.com
Oct. 31 – Nov. 1	Practical Spirometry Certification Course; **Rochester, MN**	Mayo Pulmonary Services Education Office, (800) 533-1653



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CRCE through the Journal, a program for American Association for Respiratory Care (AARC) members to gain credit for continuing education, is now in its thirteenth year. By reading RESPIRATORY CARE—the science journal for respiratory care professionals—and completing this examination, AARC members may earn credit for continuing education.*

This 50-item, multiple-choice examination is based on papers published from July 2001 through June 2002 in RESPIRATORY CARE. The issue and page numbers of the paper on which a question is based are shown in brackets following the question. You may consult the cited paper; however, we encourage you to read the paper in its entirety before answering the question. Choose the single most-correct answer, and mark the answer sheet, which is located following Page 944.

Mail your completed answer sheet by September 30, 2002. Answer sheets postmarked after September 30, 2002 will not be processed. The Answer Key for CRCE through the Journal will be published in the October issue of RESPIRATORY CARE. AARC members can access their CRCE transcripts via the AARC's Web site: http://www.aarc.org. Your CRCE through the Journal results will be available as soon as your answer sheet is scored and the score posted to your transcript.

We are indebted to H Fred Hill MA RRT, Timothy B Op't Holt EdD RRT FAARC, William Pruitt MBA RRT, and William Wojciechowski MS RRT, faculty members in the Department of Cardiorespiratory Care at the University of South Alabama, Mobile, Alabama.

*The acceptance of these credits for the fulfillment of license-mandated continuing education is dictated solely by the licensure law of each individual state.

QUESTIONS:

Please follow the instructions on the previous page, and record your answers on the perforated form provided following Page xxx.

1. In the study by Chatmongkolchart et al, what was the trigger variable for all the ventilators used in the study?

   a. time
   b. flow
   c. pressure
   d. volume

   [July 2001;46(7):666-677]

2. Based on the data gathered by Hoberty et al, what was the most important criterion reported for establishing a staff-to-patient ratio?

   a. severity of pulmonary disease
   b. availability of space or equipment
   c. presence of cardiac problems
   d. safety

   [July 2001;46(7):694-697]

3. Which of the following statements comprise the definition of an acute exacerbation of idiopathic pulmonary fibrosis, proposed by Kondok et al and used in the case report written by Nishiyama et al?

   I. the presence of newly developing diffuse pulmonary infiltrates
   II. the absence of apparent infectious agents
   III. worsening hypoxemia
   IV. exacerbation of dyspnea within a few weeks

   a. I and IV only
   b. II and III only
   c. I, III, and IV only
   d. I, II, III, IV

   [July 2001;46(7):698-701]

4. According to Muhammad et al, what is the median age for the development of granular cell tumors?

   a. 30 to 40 years old
   b. 40 to 50 years old
   c. 50 to 60 years old
   d. 60 to 70 years old

   [July 2001;46(7):702-704]

5. In the study by Schultz et al, what conclusions did the authors make?

   a. that protocol-directed weaning resulted in a longer weaning time than physician-directed weaning
   b. that both weaning approaches were equivalent
   c. that physician-directed weaning resulted in a longer weaning time than protocol-directed weaning
   d. that any difference between the two weaning techniques was based on the patient's diagnosis

   [August 2001;46(8):772-782]

6. According to Cordero et al, comparing endotracheal suctioning frequencies in very-low-birthweight (VLBW) mechanically ventilated infants, which of the following outcome variables are accurate?

   I. VLBW infants suctioned every 4 hours had a greater incidence of ventilator-associated pneumonia.
   II. The number of reintubations for the two groups was essentially the same.
   III. More of VLBW infants who were suctioned every 8 hours were discharged home on oxygen, compared to the VLBW group suctioned every 4 hours.
   IV. Bloodstream infection was greater in the VLBW group of infants suctioned every 8 hours, compared with that for the VLBW infant group suctioned every 4 hours.

   a. I and III only
   b. II and IV only
   c. II, III, and IV only
   d. I, II, III, IV

   [August 2001;46(8):783-788]

7. What conclusions were drawn from the study on mechanical ventilation reliability performed by Blanch?

I. The more a ventilator is used, the more reliable it becomes.
II. Previous repairs tend to decrease a ventilator's reliability.
III. Any brand of ventilator tends to be more reliable when it is first introduced than when it has been on the market longer.
IV. Ventilator reliability is the same throughout the "life" of the brand.

a. I only
b. I and II only
c. II and IV only
d. I, II, and III only

[August 2001;46(8):789-797]

8. In the GOLD Executive Summary, what are the components of COPD management?

I. assess and monitor the disease
II. reduce the risk factors responsible for COPD
III. manage stable COPD
IV. manage exacerbations of COPD

a. I and IV only
b. I, II, and III only
c. II, III, and IV only
d. I, II, III, IV

[August 2001;46(8):798-825]

9. According to Na Han et al, what are the Centers for Disease Control criteria for nosocomial pneumonia?

I. rales or dullness to percussion on physical examination of the chest
II. progressive lung consolidation on chest radiography
III. change in sputum characteristics from mucoid to purulent
IV. refractory hypoxemia

a. I and IV only
b. I, II, and III only
c. II, III, and IV only
d. I, II, III, IV

[September 2001;46(9):891-896]

10. According to Na Han et al, what was the difference in the percent mortality of patients receiving mechanical ventilation with circuit change intervals of 2 days and 7 days?

a. No difference was found.
b. Patients whose ventilator circuits were changed every 7 days had a lower mortality.
c. Patients whose ventilator circuits were changed every 2 days had a lower mortality.
d. The data between the 2-day-change and 7-day-change groups were inconclusive.

[September 2001;46(9):891-896]

11. What were the results of the study conducted by Kuluz et al concerning the F_{IO_2} in infants receiving O_2 via a nasal cannula?

a. The F_{IO_2} decreased as an infant's respiratory rate increased.
b. Infants received a safe level of F_{IO_2} regardless of the liter flow of O_2 to the nasal cannula.
c. Infants receiving O_2 via a nasal cannula at < 2 L/min are at risk for hyperoxic lung injury.
d. No correlation was found between the F_{IO_2} received by infants wearing a nasal cannula and the O_2 liter flow.

[September 2001;46(9):897-901]

12. The $\dot{V}_{O_2}$ measurements obtained by Mitsuoka, et al, in their study of the utility of measuring the O_2 cost of breathing were expressed as a percentage of what value?

a. the baseline $\dot{V}_{O_2}$ value
b. the $\dot{V}_{O_2}$ measured at the onset of data collection following the 5-minute adjustment period
c. the $\dot{V}_{O_2}$ obtained after the onset of the data collection following the 5-minute adjustment period
d. the $\dot{V}_{O_2}$ measured at the end of the weaning trial

[September 2001;46(9):902-910]

13. Based on the article by Dawn et al, what are the advantages of multidetector-row spiral CT scanners in thoracic imaging?

    I. Multidetector-row spiral CT (MDSCT) scanners are faster than conventional CT scanners.
    II. MDSCT scanners provide better volumetric data acquisition in thoracic imaging than conventional CT scanners.
    III. Conventional CT scanners use a continuously rotating gantry and continuous table movement.
    IV. MDSCT scanners require a longer breath-hold time than conventional CT scanners.

    a. I only
    b. I and II only
    c. III and IV only
    d. I, II, and IV only
    [September 2001;46(9):912-921]

14. Foland et al listed several advantages for using APRV. Which of the following is included as an advantage for APRV?

    a. Sedation and paralytic agents are used with APRV to reduce oxygen consumption.
    b. Spontaneous breathing is allowed in between mandatory positive pressure breaths.
    c. APRV is able to ventilate the patient at a lower peak airway pressure.
    d. APRV is well established in the management of pediatric patients receiving mechanical ventilation who have asthma.
    [October 2001;46(10):1019-1023]

15. According to Foland et al, in establishing the settings for APRV, which of the following describes the variables that most influence the creation of intrinsic PEEP?

    a. release time and low pressure
    b. rate and inspiratory time
    c. PEEP and total cycle time
    d. inspiratory flow and rate
    [October 2001;46(10):1019-1023]

16. According to Kallet et al, which of the following factors may be considered in making corrections to the initial tidal volume based on predicted body weight?

    a. From an initial setting of 6 mL/kg, tidal volume can be reduced in increments of 0.5 mL/kg over 8 to 10 hours to reach a target of 4 mL/kg if necessary.
    b. Tidal volumes may be increased by 0.5 mL/kg if P_{aCO_2} is greater than 65 mm Hg.
    c. Corrections may be made for age when the patient is over 70 years old.
    d. Adjustments may be made to account for compression volume loss and to avoid nuisance adjustments.
    [October 2001;46(10):1024-1037]

17. According to Kallet et al, which of the following describes the point at which weaning begins in the ARDS Network protocol?

    I. The patient is exhibiting inspiratory effort.
    II. Neuromuscular blockade has been removed.
    III. The patient is tolerating 2 hour trials of CPAP at 5 cm H_2O.
    IV. PEEP is < 8 cm H_2O and oxygenation is stable on F_{IO_2} of < 0.40.

    a. I and III only
    b. II and IV only
    c. I, II, and IV only
    d. I, II, and III only
    [October 2001;46(10):1024-1037]

18. According to Montori and Guyatt, why has the practice of medicine been so slow in responding to strong, definitive support to change its approaches and treatment?

    I. There has been considerable variability in expert opinion.
    II. Recommendations in textbooks and reviews have lagged behind the evidence.
    III. Financial concerns about cost have caused delays.
    IV. Concerns about lawsuits for malpractice have forced extreme caution in making any change in the status quo.

a. I and II only
b. III and IV only
c. II and III only
d. I and IV only.
[November 2001;46(11):1201-1211]

19. According to Montori and Guyatt, which of the following should have the greatest impact on clinical decision-making?

a. physiologic studies
b. randomized controlled trials
c. *n* of 1 randomized trials
d. observational studies
[November 2001:46(11):1201-1211]

20. According to Hess, in the article on the evidence for secretion clearance techniques, the author concluded that:

a. There has been a recent increase in high-level evidence to support only 2 of the 15 secretion clearance techniques listed in the MEDLINE review of the topic.
b. Chest physiotherapy has a significant impact on the long-term outcome and quality of life in patients with CF as shown by the evidence reviewed.
c. Chest percussion and vibration showed a clear benefit compared to postural drainage alone.
d. Many of the studies from the MEDLINE review of the topic were small and used a crossover design.
[November 2001;46(11):1276-1292]

21. According to MacIntyre's article on COPD management, putting together a review of the evidence has several limitations. Which of the following is one of those limitations?

a. Absence of proof is not proof of absence.
b. There is a lack of clinical studies derived from randomized control trials.
c. The review included bench data and observational experience.
d. The role of surgery in managing COPD was included but not part of the meta-analyses.
[November 2001:46(11):1294-1303]

22. According to Meade and Herridge, how did the "open lung" protocol deal with patient-ventilator disconnections?

a. The patient was hyperventilated with a manual resuscitator bag with PEEP and 100% oxygen prior to and after disconnections.
b. The patient was given 4-6 mandatory breaths triggered by the therapist after any disconnection.
c. PEEP was increased by 4 cm H_2O for 10 minutes after any disconnection.
d. The lungs were inflated to a pressure of 35-40 cm H_20 and held for 40 seconds after any disconnection.
[December 2001:46(12):1368-1376]

23. According to Rowe et al, which of the following conclusions were made concerning oral corticosteriods?

a. Patients receiving oral corticosteriods were similar to the control group in their need for $ß_2$ agonists post-emergency care.
b. The associated number of patients needed to treat to prevent 1 relapse after an asthma exacerbation was 18.
c. Patients with questionable compliance or inability to afford oral steroids may need to be treated with intramuscular corticosteriods post-emergency care.
d. There was no significant difference between the corticosteriod group and the control group regarding relapse during the first week post-emergency care.
December 2001:46(12):1380-1390]

24. Which of the following are Grade B recommendations about ventilator-associated pneumonia (VAP) based on information found in the article on evidenced-based airway management by Littlewood and Durbin?

I. Selective digestive decontamination in conjunction with systemic prophylactic systemic antibiotic treatment should be used in high-risk patients.
II. Noninvasive ventilation is preferred over intubation and mechanical ventilation.
III. An identified person or group should have responsibility for monitoring nosocomial VAP rates.
IV. Heat and moisture exchangers with bacterial filtering capability are an effective strategy to reduce the incidence of VAP.

a. I and II only
b. II and III only
c. III and IV only
d. II and IV only

[December 2001:46(12):1392-1405]

25. According to Heitman and Flemons, which of the following is true concerning changes in a patient's weight?

a. Weight gain of 10 pounds or more in patients with mild or no obstructive sleep apnea (OSA) is predictive of a 32% increase in the apnea-hypopnea index (AHI).
b. A 10% (±) weight change has at least a 25% effect, positive or negative, in the AHI.
c. A 6-fold increase in the odds of developing moderate to severe OSA has been linked to 25% weight gain in patients with mild or no diagnosis of (OSA).
d. Weight loss along with a 15 ° elevation in the head of the bed has been found to reduce AHI significantly in patients with mild to severe OSA.

[December 2001:46(12):1418-1432]

26. Which of the following statements is FALSE concerning the administration of albuterol to intubated newborns, according to a survey by Ballard et al?

a. 80% of the respondents use a metered-dose inhaler to deliver albuterol to intubated newborns.
b. The usual dose of nebulized albuterol was 1.25-2.5 mg.
c. When nebulizing albuterol, a majority of respondents place the nebulizer adjacent to the Y-piece of the ventilator circuit.
d. When administering albuterol via MDI, most respondents administer 1-2 actuations.

[January 2002;47(1):31-38.]

27. According to Heulitt et al, what is the effect on work of breathing (WOB) during partial liquid ventilation (PLV) in spontaneously breathing animals?

a. In animals with induced ARDS, WOB decreases significantly during PLV.
b. In healthy animals, WOB is acceptable during PLV.
c. PSV decreases the WOB at any liquid level better than it does with gas ventilation.
d. Resistive WOB increases significantly, while elastic WOB decreases significantly with the use of PLV.

[January 2002;47(1):39-47]

28. In the study of pulse oximetry errors when perfusion was low, or when there was motion artifact, Gehring et al found that:

a. There is a wide variation among the pulse oximeters tested in displayed S_{pO_2} from actual S_{pO_2} when the subject's perfusion was decreased by a compression balloon.
b. There is a wide variation among the pulse oximeters tested in displayed pulse from actual pulse when the subject scratched or tapped.
c. Even in the presence of decreased perfusion or motion, the pulse oximeters tested reliably measured S_{pO_2}.
d. In the presence of motion and decreased perfusion all tested pulse oximeters reliably measured pulse.

[January 2002;47(1):48-60]

29. According to the Evidence-Based Guidelines for Weaning and Discontinuing Ventilatory Support, a positive outcome for which of the following procedures will likely result in successful ventilator discontinuation?

a. check of the patient's level of consciousness
b. spontaneous breathing trial
c. maximum inspiratory pressure measurement
d. tidal volume measurement
[January 2002;47(1):69-90]

30. A patient has received propofol to maintain comfort and maintain patient-ventilator synchrony. Which of the following should the therapist expect 30 minutes following discontinuation of the propofol, according to Khamiees et al?

a. The patient should be ready for extubation.
b. Extubation may not be immediately successful.
c. The rapid shallow breathing index should be <100.
d. A normal pattern of ventilation.
[February 2002;47(2):150-153]

31. A small clinical trial by Shortall et al, comparing an oral/MDI regimen versus an I.V./aerosol regimen for the treatment of exacerbation of COPD revealed:

a. a significant decrease in length of stay with the oral/MDI regimen
b. a significant increase in FEV_1 with the I.V./aerosol regimen
c. no significant differences in outcomes variables
d. more frequent treatment failures with the oral/MDI regimen
[February 2002;47(2):154-158]

32. In a study of two forms of ipratropium bromide (metered-dose versus dry powder), Cuvelier et al found that:

a. Use of the dry powder inhaler (DPI) resulted in a significantly higher FEV_1 compared to the MDI form.
b. The persistence of response was greater with the MDI form compared to the DPI form.
c. There were no side effects from either form.
d. Patients thought it was easier to handle the DPI versus the MDI.
[February 2002;47(2):159-166]

33. A patient with asthma is being treated in the emergency department (ED). Following 3 treatments with 2.5 mg albuterol by aerosol, the therapist observes the following:

PEF	150 L/min (Pred = 450 L/min)
P_{aCO_2}	45 mm Hg
Sensorium	drowsy

What should the therapist recommend, according to Hallstrand and Fahy?

a. discharge from the ED with a physician's office visit in 24 hours
b. admission to a general medical ward with frequent treatments
c. two additional treatments in the ED
d. admission to the intensive care unit
[February 2002;47(2):171-182]

34. A patient with an exacerbation of COPD arrives in the emergency department (ED) with a respiratory rate of 35 breaths/minute, accessory muscle use, a pH of 7.32 with a P_{aCO_2} of 56 mm Hg. He is quite somnolent. According to the recommendations by Pierson, the respiratory therapist should recommend which of the following?

a. intubation and mechanical ventilation
b. noninvasive positive pressure ventilation
c. intubation and oxygenation adminstration with a Briggs adapter
d. three successive 2.5 mg. albuterol treatments
[March 2002;47(3):249-262]

35. To assure that excessive lung stretch is avoided, MacIntyre advocates a tidal volume of ___ mL/kg ideal body weight in patients with substantial lung injury.

a. 12
b. 10
c. 8
d. 6
[March 2002;47(3):266-274]

36. What have Saura and Blanch concluded about the selection of the best combination of PEEP and tidal volume to recruit alveoli in ARDS?

a. Most alveoli are recruitable by PEEP and low tidal volume.
b. Selection of the best PEEP and tidal volume remain controversial.
c. Most alveoli are recruitable with a high tidal volume and low PEEP.
d. All alveoli are recruitable with the right combination of PEEP and tidal volume.

[March 2002;47(3):279-292]

37. According to Hess and Bigatello, lung recruitment maneuvers in ARDS:

I. may decrease atelectasis
II. may cause hypotension and barotrauma
III. improve patient survival
IV. are applied using a recognized standard methodology

a. I and II only
b. III and IV only
c. II, III, and V only
d. I, II, and III only

[March 2002;47(3):308-317]

38. According to Campbell and Davis, current recommendations for an optimal "lung-protective" approach in mechanical ventilation of patients with ARDS and acute lung injury include limiting V_T to _____ and limiting transpulmonary pressure to _____.

a. 3 mL/kg; 20 cm H_2O
b. 6 mL/kg; 25 cm H_2O
c. 8 mL/kg; 30 cm H_2O
d. 6 mL/kg; 35 cm H_2O

[April 2002;47(4):416-426]

39. In general, a spontaneous breathing trial (SBT) should be of _____ minutes duration, according to Epstein.

a. 30
b. 60
c. 120
d. 240

[April 2002;47(4):454-468]

40. Which of the following statements regarding resistance to airflow imposed by a tracheostomy tube is false, according to Jaeger et al?

a. The larger the diameter, the lower the resistance.
b. The shorter the tube, the lower the resistance.
c. Irregular walls (tube material or secretions) lower the resistance.
d. Curves in the tube increase the resistance.

[April 2002;47(4):469-482]

41. A higher risk of extubation failure was noted by Epstein for a hemoglobin < _____ mg/dL.

a. 20
b. 15
c. 10
d. 5

[April 2002;47(4):483-495]

42. According to MacIntyre, the most effective drugs for patient anxiety in the ICU are:

a. the benzodiazepines
b. the barbiturates
c. the analgesics
d. the narcotic antagonists

[April 2002; 47(4):508-518]

43. In a study of 10 respiratory therapy programs, which of the strategies intended to develop critical thinking and decision-making was least utilized, according to Hill?

a. role modeling by teacher
b. computer-assisted instruction
c. problem-based learning
d. clinical patient rounds

[May 2002:47(5):571-577]

44. According to Stoller et al, what was the most common reason for spirometry to be performed at bedside rather than in the pulmonary function laboratory for hospitalized patients at the Cleveland Clinical Hospital?

a. late admission
b. unsafe to transport patient
c. patient in intensive care
d. chest tube in place

[May 2002;47(5):578-582]

45. Which of the following statements is false when comparing bedside spirometry to laboratory pulmonary function testing, according to Stoller et al?

a. Most orders for bedside spirometry are not justified by clinical circumstances.
b. Implementation of a "quality program" resulted in a greater percentage of acceptable spirograms from bedside testing than was achieved in the pulmonary function laboratory.
c. The study did not demonstrate a difference in technical performance based on numbers of tests (< 10 vs ≥ 10) performed by each respiratory therapist at bedside.
d. The rate of achieving high-quality bedside spirograms was enhanced by the "quality program."

[May 2002;47(5):578-582]

46. According to Wilkins et al, which skill in patient assessment was highest in physicians' expectations of respiratory therapists?

a. interpret pulmonary function tests
b. interpret arterial blood gases
c. assess vital signs
d. assess lung sounds

[May 2002;47(5):583-585]

47. According to Foland et al, most pediatric physicians will give steroids as prophylaxis for airway edema for "air leak" values of _____ cm H_2O or less.

a. 20
b. 25
c. 30
d. 40

[June 2002;47(6):662-666]

48. Which of the following patient aspects of a study (by Granton et al) of noninvasive ventilatory support for stable cystic fibrosis patients failed to improve?

a. REM or Stage 4 sleep time
b. minute ventilation
c. respiratory rate
d. pleural pressure swings

[June 2002;47(6):675-681]

49. A study by Meyers et al of the Neotrend blood gas sensor revealed:

I. ease of use
II. reasonable degree of accuracy
III. safety with the device
IV. cost saving

a. I and IV only
b. II and III only
c. II, III, and IV only
d. I, II, III, IV

[June 2002;47(6):682-687]

50. The Glenn shunt is a palliative procedure intended for the specific heart defect:

a. transposition of the great vessels
b. tricuspid atresia
c. coarctation of the aorta
d. ventricular septal defect

[June 2002;47(6):688-692]

Make sure to double check your work and to mark the answer sheet clearly. Mail your completed answer sheet by September 30, 2002.

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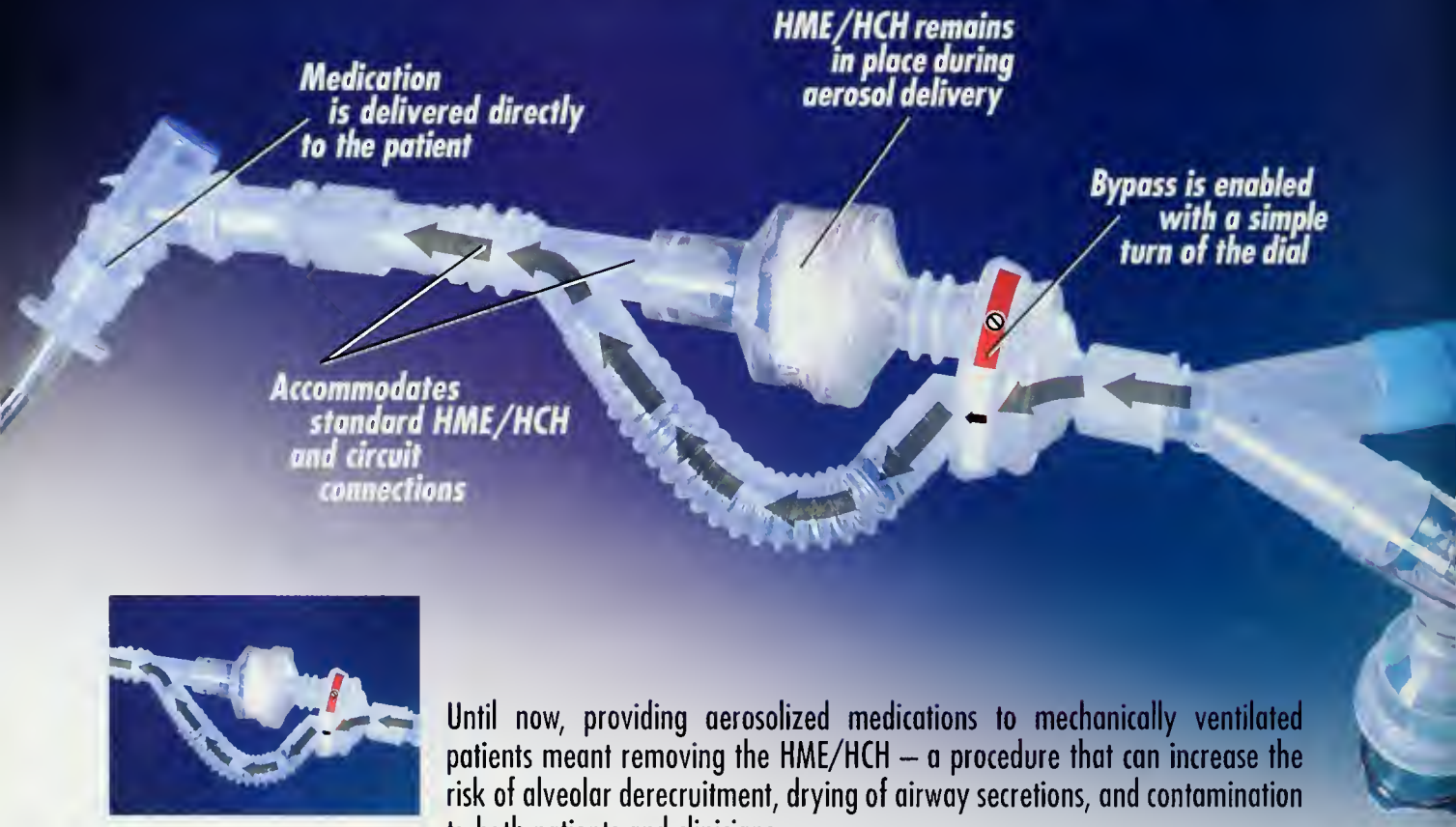
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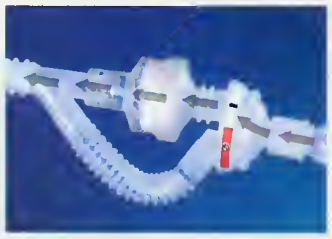

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